A PROSPECTIVE OPEN LABELLED
NON RANDOMIZED PHASE-II CLINICAL TRIAL OF
"KADUKKAI CHOORANAM FOR

"AKKINI SELATHUMAM"

(DIABETIC NEUROPATHY)

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A PROSPECTIVE OPEN LABELLED NON RANDOMIZED PHASE-II CLINICAL TRIAL OF "KADUKKAI CHOORANAM FOR AKKINI SELATHUMAM (DIABETIC NEUROPATHY)"** is abonafide work done by **Dr.RAJENDRAM AJANTHAN (Reg. No.321611008)** Govt. Siddha Medical College, Palayamkottai - 627 002 in partial fulfilment of the university rules and regulations for award for **MD (S) POTHU MARUTHUVAM (BRANCH-I**) under my guidance and supervision during the academic **year OCTOBER 2016-2019.**

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PLAGIARISM CERTIFICATE

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DECLARATION BY THE CANDIDATE

I, Dr.**R.Ajanthan** declare that the dissertation entitled **"A PROSPECTIVE OPEN LABELLED NON RANDOMIZED PHASE-II CLINICAL TRIAL OF "KADUKKAI CHOORANAM FOR AKKINI SELATHUMAM (DIABETIC NEUROPATHY)''**submitted for the degree of MD Siddha Medicine of Government Siddha Medical College, Palayamkottai, Tirunelveli, Tamil Nadu(The Tamil Nadu Dr. M.G.R. Medical University, Chennai) the record of work carried out by me under the supervision and guidance of **Prof. Dr.A.Manoharan, MD(S),(Ph.D).** Head, Department of Pothu Maruthuvam, Govt. Siddha Medical College, Palayamkottai. This work has not formed the basis of award of any degree, diploma, associateship, fellowship or other titles in the university or any other university or institution of higher learning.

Signature of the candidate

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Place : Palayamkottai

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ABBREVIATIONS

ADA	-	American Diabetes Association
ATP III	-	Adult Treatment Panel III
AMORIS	-	Apo lipoprotein-Related Mortality Risk
BAI	-	Body Adiposity Index
BMI	-	Body Mass Index
CHD	-	Coronary Heart Disease
CETP	-	Cholesteryl Ester Transfer Protein
CD	-	Cluster of Differentiation
CVD	-	Cardiovascular Disease
CRP	-	C-reactive protein
DM	-	Diabetic Mellitus
DCCT	-	Diabetes Control and Complication Trial
EDIC	-	Epidemiology of Diabetes Intervention and Complication
FFA	-	Free Fatty Acid
GAD	-	Glutamic-acid-decarboxylase
HBAIC	-	Glycated Haemoglobin
HSL	-	Hormone-sensitive Lipase
HDL-C	-	High Density Lipoprotein Cholesterol
HC	-	Hip Circumference
IDF	-	International Diabetic Federation
IL-6	-	Interleukin 6
IGT	-	Impaired Glucose Tolerance
IDDM	-	Insulin Dependent Diabetes Mellitus
LDL-C	-	Low Density Lipoprotein Cholesterol
LPL	-	Lipoprotein Lipase
NIDDM	-	Non-Insulin Dependent Diabetes Mellitus
NHDL-C	-	Non High Density Lipoprotein Cholesterol
NGSP	-	National Glycohaemoglobin Standardisation Programme
NCEP	-	National Cholesterol Education Program
OGTT	-	Oral Glucose Tolerance Test
TG	-	Triglycerides
TC	-	Total Cholesterol

T2DM	-	Type-II Diabetes Mellitus
UKPDS	-	United Kingdom Prospective Diabetes Study
VAI	-	Visceral Adiposity Index
VLDL-C	-	Very Low Density Lipoprotein Cholesterol
VAT	-	Visceral Adipose Tissue
W.H.O.	-	World Health Organization
NCS	-	Nerve Conduction Study
KC	-	Kadukkai Chooranam
DN	-	Diabetic Neuropathy

ABSTRACT

Diabetes Mellitus is one of the most prevalent and serious metabolic diseases in the world which is predicted to increase dramatically. Diabetes is frequently associated with longterm complications with macrovascular and microvascular origin. Diabetic neuropathy AKKINI SELATHUMAM) is one of the most common complications of diabetes mellitus. It is the most common neuropathy; it is estimated that about 50 percent of patients with diabetes mellitus will eventually develop some form of neuropathy. The study was aimed at evaluating clinical efficacy of the herbal formulation of KADUKKAI CHOORANAM (KC) on AKKINI SELATHUMAM in patients with type-II diabetes. This study is an open labelled non randomized Phase-II clinical trial spanning 90 days. About 40 subjects of age range between 40-70 years. In all 40 diabetic patients who were under treatment were randomly sampled for the study. Socio- demographic data were collected using predesigned questionnaires. Glycated haemoglobin levels, lipid profile, HBA1-C, fasting blood sugar and other haematological investigations were estimated using standard procedures before and after treatment. Diabetic Neuropathy was defined as per Use of the leeds assessment of neuropathic symptoms and signs pain scale. The Statistical analysis was done by SPSS statistical package version 20.0. Paired 2 tailed test revealed that the fasting ($P<0.0001$) and postprandial blood glucose (0.0001) and HbA1c ($P<0.0001$), in lipid profile TC ($p<0.0001$), LDL ($p<0.0001$), HDL (<0.0001) and TGL ($p<0.0001$) showed significant reduction after Kadukkai chooranam intervention. The liver, renal functions along with the haematological parameters were well within the normal range. The trial drug subjected to biochemical and pharmacological studies and gave significant results also. The results KC to be beneficial for the treatment of Akkini selathumam (Diabetic Neuropathy) in type-II diabetes; further follow-up studies are warranted to confirm the safety aspects of kadukkai Chooranam use.

CHAPTER-I
INTRODUCTION

1.1BACKGROUND

The Siddha system is one of the ancient and Spirituality enriched traditional medical system of india. The fundamentals of Siddha system of medicine is to strength the body and mind. The funtamentals of treatment is to neutrolize the three vital humours. In the Siddha system of medicine is to formed creation and genesis of matter on earth are controlled and regulated by the Pancha bhoothams, and it based on thridoshas and dasa naadigal at microcosm and macrocosm plane, an imbalance in the creative forces subsequently causes defective function affecting the existence, qualitatively and quantitatively. Siddha system always tries to treat the patient, not the disease and hence treatments for the same disease may vary from individual to individual.Hyperlipidemia is a more prevalence and incidence in 80% to 88% (Sarit et al 2016) with approximately 40% - 48% is more incidence in middle age group.

According to the classical text Noi nadal and Noi Mudhal Nadal Part II and Yugi Vaidhaya Chinthamani 800 are clearly discribed that preclinical symptoms of Akkini Selathumam (Diabetic Neuropathy).

Our ancestors elaborated the knowledge about Neerizhivu Noi. Theraiyar in "Therankarisal" has mentioned the classification about the diseases, the urinary system into two major categories of "Neerinaiperukkal (Polyurea)" and Neerinai arukkal Noi (Oliguria).According to Noi nadal book it was classified accoding to frequency, specific gravity and frothy urination. Diabetes mellitus has to comes under Neerinaiperukkal (Polyurea),and Neerinai arukkal Noi (Oliguria) is coming under categories of complicalication of diabetic related disease. According to clinical symptoms and envagai thervugal Neerizhivu noi is a main primary criteria for classified in Vali, Azhal and Iyam.

As per the Siddha perception classical text Noinadal and NoiMudhalNadal Part II and Yugi Vaidhaya Chinthamani 800 are clearly mentioned that preclinical symptoms of Akkini Selathumam.The main symptoms and signs are burning sensation of the upper and lower limb, Polyurea ,Polyphagia ,Polydepsia and Polypepsia, Cough with expectoration, Slight fever and chills.

Diabetes Mellitus is one of the most prevalent and serious metabolic diseases in the world which is predicted to increase dramatically. It is frequently associated with long-term complications with macro vascular and micro vascular origin. Diabetic neuropathy is one of the main neurological complications in diabetic disease (Diabetic Poly neuropathy.

Diabetic neuropathy is the most prevalent chronic complications of diabetes. This heterogeneous group of conditions affects different parts of the nervous system and presents with diverse clinical manifestations. The early recognition and appropriate management of neuropathy in the patient with diabetes is important for a number of reasons (American Diabetes Association.2017)

1. Diabetic neuropathy is a diagnosis of exclusion. Non diabetic neuropathies may be present in patients with diabetes and may be treatable by specific measures.
2. A number of treatment options exist for symptomatic diabetic neuropathy.
3. Up to 50% of diabetic peripheral neuropathies may be asymptomatic. If not recognized and if preventive foot care is not implemented, patients are at risk for injuries to their insensate feet.
4. Recognition and treatment of autonomic neuropathy may improve symptoms, reduce sequelae, and improve quality of life.

Among the various forms of diabetic neuropathy, distal symmetric polyneuropathy (DSPN) and diabetic autonomic neuropathies, particularly Cardiovascular Autonomic Neuropathy (CAN). Patients with prediabetes may also develop neuropathies that are similar to diabetic neuropathy due to poor control of diabetes. The screening for symptoms and signs of diabetic neuropathy is critical in clinical practice, as it may detect the earliest stages of neuropathy, enabling early intervention. Although screening for rarer atypical forms of diabetic neuropathy may be warranted, DSPN and autonomic neuropathy are the most common forms encountered in practice. The strongest available evidence regarding treatment pertains to these forms.

Prevention of diabetic neuropathies focuses on glucose control and lifestyle modifications. Available evidence pertains only to DSPN and CAN, and most of the large trials that have evaluated the effect of glucose control on the risk of complications have included DSPN and CAN as secondary outcomes or as post hoc analyses rather than as primary outcomes

Kadukkai Chooranam along is herbal Siddha formulation taken from the classical Siddha literatures "Gunapadam Mooligai Muthal Paakam(Page; 313) and Kadukkai Vallaraiyin Thanimanbu (Page;09) drug had been chosen to study its efficacy in the management of Akkini Selathumam. The literature collections have been proved the individual constituents of the preparation possess hypoglycemic, hypolipidemic, analgesic and anti – oxidant activities.

The above mentioned references and the pharmacological research works undergone on the constituents of the trial medicines kadukkai chooranam justified its potential effect in the clinical study of the management of Diabetic Neuropathy conditions.

RATIONALE

Kadukkai Chooranam is the single herbal formulation taken from the classical siddha literature.The drug has been chosen to study it was moer efficacy in management of Akkini Selathumam. Recently, the plant is of great interest to researchers across the global level because of its reported medicinal properties like Hypoglycemic, Antioxidant, Anti mutagenic, Acetylcholine inhibition, Anti anaphylaxis, Hepatoprotective, Anti bacterial, Hypolipidemic activities.

The above mentioned references and the pharmacological research works undergone on the constituents of the trial medicine Kadukkai Chooranam for the management of Diabetic Neuropathy .So, the trial medicine will be safe to management of Diabetic Neuropathy in Type II patient.

1.2 AIM AND OBJECTIVES

AIM

A prospective open labeled Phase – II Non - Randomized clinical study of Kadukkai Chooranam internally for Akkini Selathumam (Diabetic Neuropathy).

OBJECTIVES

A. **Primary Objective:**

To evaluate the therapeutic efficacy of Clinical Trial drugs in Akkini Selathumam

B. **Secondary Objectives:**

a. To find out the Antidiabetic, hypolipidemic,and Analgesic Pharmacological activity of Kadukkai Chooranam.
b. To determine the addition of effects and siddha parameters changes in Akkini Selathumam.
c. Study about the prevalence of Akkini Selathumam in Relation between diet and lifestyle.
d. To carried out Siddha and Modern parametric changes in Diabetic Neuropathypatient.
e. To adjudge the Antimicrobial, Physico,Phytochemical analysis of the clinical drug
f. To assessed contents of chemical compound through FTIR and SEM analytic methods.
g. To disscuss about treatment and prognosis effect after end of study.

1.3 JUSTIFICATION OF RESEARCH

Apart from classical risk factors like diabetic dyslipidemia, elevated HbA1C has now been identified as an independent risk factor for cardiovascular disease in subjects with or without diabetes. Estimated risk of cardiovascular disease has shown to be increased by 18% for each 1% increase in absolute HbA1C value in diabetic population. Although new tests of glycation are now available, they are not without

limitations of their own, very expensive and impractical for everyday use in developing countries like India.

This study was an attempt to reassess the association of glycemic control and lipid profile using HbA1C whiles taking into consideration some of the factors affecting its use. This study was also afford health care providers attending to diabetic patients, the needed information as to when to use HbA1C in clinical monitoring of glycation and diabetic dyslipidemia.

Now a days world turned to herbal medicines for maximum reduce the risk factors from chemcials. Therefore these are simple and efficient herbal formulations treated in diabetic dyslipidemia with out adverse / side effects.

CHAPTER-II

REVIEW OF LITERATURE

2.1.1IN JOURNAL:

The various journals to collections related to kadukkai *(Terminalia chebula)*

Fig. 2;1Kadukkai

Fig.2;2 Kadukkai Chooranam

Taxonomy •

- Kingdom: Plantae-Plants
- Subkingdom: Tracheobionta-Vascular plants
- Super division: Spermatophyta-seed plants
- Division: Magnoliophyte- flowering plants
- Class: Magnoliopsida dicotyledons
- Subclass: Rosidae
- Order: Myrtales
- Genus: Terminalia
- Species: Terminalia chebula.

Macroscopic characters:

It is a deciduous tree, younger stems glabrescent and woody. These are 10 – 20 cm long, sub – opposite, simple; exstipulate; petiolate; laminae broadly elliptic to elliptic oblong, rarely ovate, the bases obtuse, the margins entire, the tips acute, glabrescent. Resin and a purgative principle of the nature of anthraxquinone and sennoside are also present. These are single, rough, ellipsoid, 1.0-2.0 cm by 0.2 -0.7 cm and without ridges.

Microscopic characteristics:

Transverse section of the fruit shows epicarp composed of a layer of epidermal cells, the outer tangential wall and upper portion of the thick radial walls. Testa, one layer of large cubical cells, followed by a zone of reticulates parenchyma and vessel; tegument consists of collapsed parenchyma. Cotyledon folded and containing aleurone grains, oil globules and some rosette aggregate crystals. The powder of the plant is brown in color, shows a few fibers under the microscope, vessels with simple pits and groups of sclereids (Aneja Kr,2010).

Chemical constituents:

Total phyto-constituents of Terminalia chebula are hydrolysable tannins (which may vary from 20-50%) and they are responsible for pharmacological activities. The tannin content varies with the geological variation. Flavonol glycosides, tri-terpenoids, and coumarin conjugated compounds with gallic acid called as chebulin, and also phenolic compounds are isolated. The major eight compounds are present in the kadukkai. Gallic acid, methyl gallate, ethyl gallate, chebulagic acid, tetra-O-galloyl-β-D-glucose, and ellagic acid, chebulinic acid and penta-O galloyl-β-D-glucose from Terminalia chebula were isolated and checked out the reverse phase chromatography. There are seven varieties of Terminalia chebula all of which are more or less used in similar fashion but vary in specific usages and quality (Baliga MS,2010).

Pharmacological screening:

The various studies determine Terminalia chebula is one of the most ingenious plants having a wide range of pharmacological and medicinal activities. This multifaceted medicinal plant is the distinctive source of various types of compounds having various chemical structures. Though it has a number of pharmacological activities due to the presence of various types of bioactive compounds, little work has been done on the probable medicinal applications of this plant against the diseases particularly on Hypoglycemic and hypolipedimic activites.

The extracts of Terminalia chebula have been widely investigated for its various pharmacological effects due to which a number of therapeutic uses have been

associated with the plant. Terminalia chebula has been noted to possess potent **antioxidant** (SarmisthaSaha etal,2014) **,** the properties due to the presence of the phenolic compounds present in its extract. The aqueous extract of the fruits of Terminalia chebula showed antioxidant activity as evident by the fact that the extract from the plant showed significantly decreased **lipid peroxidation (** Thakur CP et al and Shaila et al 1998) effects. Moreover, the antioxidant potential associated with the plant helped it in order to possess a **hepato-protective** effect. Further kadukkai was a good anti diabetes properties (NaiamoluKotesswaraRao,2006).

Suchalatha et al 2005 has done the work of Cardio productive effect in ethanolic extract of T.chebula was investigated in isoproteronol induced myocardial tissues in rat. She was reported that T.chebula extract had cardio productive effect due to thelysosomal membrane stabilization and preventing myocardial necrosis, inhibition of alteration in the heart mitocontrial ultra structure and function in the experimental rat was observed.

Anti-inflammatory and anti-arthritic activity

Mehmood.1999 was determined about aqueous extract of dried fruit of T. chebula was high inhibition of inducible nitric oxide synthesis,it is evident to **anti-inflammatory** activity in kadukkai.

Vonshak 2003 and Shinde 2011 has found Chebulagic acid is suppressed the onset and progression of collagen induced arthritis in mice.

Nair V 2010, was done the research of hydroalcoholic extract of T. chebula produced a significant inhibition of joint swelling as compared to control in both formaldehyde-induced and CFA-induced arthritis. T. chebula treatment also reduced serum TNF-α level and synovial expression of TNF-R1.

Anti oxidant and anti cancer activity:

Ponnusankar S 2011 has worked on anti-mutagenic and anti-carcinogenic activities of 70% methanolic fruit extract against several malignant cell lines. The Chebulagic acid is a potent dual inhibition effects against COX and 5-LOX. It is

good evident in **antiproliferative** activity against HCT-15, COLO-205, MDAMB-231, DU-145 and K562 cell lines and inhibit cytochrome P450.

ChenX.2011 has done the work of ***anti-oxidant*** activites in six extracts and four pure compounds of Terminalia chebula is exhibited in-vitro *antioxidant* properties of anti-lipid peroxidation, anti superoxide radical formation and DPPH and other free-radical scavenger activities in different concentration.

Antidiabetic and renoprotective activity:

Sabu MC 2002, has done pharmacological study about STZ,Alloxan induced experimental diabetic rats, he was assesed by different extract in all group of animals, 75% methanolic extract of T. chebula (100 mg/kg body weight has to reduced the blood sugar level in normal and alloxan diabetic rats significantly within 4 h. Continued daily administration of the drug produced a sustained effect.

Rao NK 2006 was determine about the chloroform extract of T. chebula seeds (100, 200 and 300 mg/kg body weight) produced dose-dependent reduction in blood glucose of diabetic rats in both short term and long term study (300 mg/kg body weight for 8 weeks). Further he noticed remarkable **renoprotective activity** of T. chebula treated rats.

Kumar GPS 2006 was found oral administration of ethanolic extract of fruits of T. chebula (200 mg/kg body weight for 30 days) reduced the levels of blood glucose and glycosylated hemoglobin in streptozotocin (STZ)-induced experimental diabetic rats.

Murali YK 2007 has done a similar study, aqueous extract of T. chebula (200 mg/kg body weight for two months) reduced the elevated blood glucose and increase in glycosylated hemoglobin. The same dose also showed a marked improvement in controlling the elevated blood lipids as well as decreased serum insulin levels. The in vitro studies with pancreatic islets showed that the insulin release was nearly two times more than that in untreated diabetic animals.

i) Kannan VR 2010 and Senthilkumar2008 was worked in streptozotocin induced diabetic rats models.At the end of study revealed the extract of Terminalia chebula

fruit and seeds was an exhibited dose dependent reduction in blood glucose and renoprotective activity .

ii) Singh I 2009 did a diabetic work streptozotocin induced rat models, 200 mg/kg of kadukkai choornam was reduced in total body weight and improved the glucose tolerance, 44% of reduction in the peak blood glucose at 2nd hour in glucose tolerance test. The fruit extract of Terminalia chebula was a significantly reduced in dose-dependent glucose lowering effect.

Phytochemistry

Jagetia GC.2002 has done Terminalia chebula contains high phenolic content, especially hydrolyzable tannins, anthraquinone, flavonol,carbohydrates, glucose and sorbitol . The triterpenes have been reported which are arjun glucoside 1, arjungenin and the chebulosides 1 and 2. Other constituents contains tannins up to 30%, chebulic acid 3-5%, chebulinic acid 30%, tannic acid 20 40%, ellagic acid, 2,4-chebulyi– β-D-gluco pyranose, gallic acid, ethyl gallate, punicalagin terflavin A, terchebin, some purgative of the nature of nthraquinone, flavonoids like luteolin, rutins, and quercetin etc

. **Anti-bacterial and Anti fungal activites**

Kannan et al.2009 has investigated on two anti-bacterial compounds, Gallic acid and ethyl ester against methicillinresistant Staphylococcus, have been isolated from ethyl alcohol extract of fruits of Terminalia chebula. Terminalia chebula is well effective against Helicobacter pylori, a bacterium responsible for gastritis, ulcer and stomach cancers.

Khanan et al 2015 was studied about resistant to Staphylococcus against methicillin , and it was well effective and sensitive to H.Pylori organism and it was effect to Stomach malignancy (Kannan et al 2009).

Jeong AHN et al 2002 have did a work of Aqueous extract in T.Chebula in **Anti fungal** activity, He found the extract was highly effective against the *Candida albicans ,Epidermophyton floccosum,Microsporumgy pseum* and *Trichophyton rubrum*.

2.1.1 **SIDDHA LITERATURE:**

According to the WHO, the herbal medicines have been defined as those containing plant parts or plant materials in raw state or processed form (Krishnan. KS 1998) containing bioactive principles, are to be considered a important form and ensured to follow the Protocol for drug research in traditional system of medicine. The Siddha system of medicine encompasses around 600 medicinal plants is described in siddha materiamedica (Gunapadam Mooligai vaguppu2016). From the abundant source of herbal preparations in different formulations are practiced more than ten decades. So, it must be ensured that the quality of the drugs should not be compromised, the efficacy of the drugs should be maximized, the adverse effects should be minimized when prepared it in a absolute protocol as mentioned by the Siddha literatures. In Tibetan literature was mentioned, the therapeutic uses in root was used for bone disease ,Stem for muscular disease ,Bark used for skin disease, and Fruits for internal organ related diseases (Pandy GS,Chunekar et al.1999).

The Kadukkaichooranam is an Internal medicine comes under the Chooranam types of Medicines. which is used in Siddha and Traditional medicine for constipation, chronic diarrhoea, gastric ulcer, gastroenteritis, asthma, cough, dyspnoea, dyspepsia, haemorrhoids, Candidiasis, parasites, malabsorption syndrome, Hepatomegaly, renal calculi, urinary discharge, tumours, skin disease, memory loss, epilepsy, diabetes, cardiovascular disease, anorexia and wounds(Nadkarni. K.M. 1976).

The "Kadukkaichooranam" is mentioned in several Siddha literature "Gunapadam Mooligaivaguppu Part -1"is indicated for Mega disorder (Diabetes), Burning sensation of upper and lower limbs (Poly neuropathy), liver diseases and anaemia.

In Siddha system of medicine is classified 4448 types of diseases. According to Yugi Vaithiya Chinthaamani-800, Mega noi (or) "neerinai perukkal noikal" is classified into 20 types.In Tamil siddha maruthuvam, the synonyms of the MadhumegaNoi(மதுமேகநோய்)are,Pramegham(பிரமேகம்),Neerilivu(நீரிழிவு)இSalaroga m(சலரோகம்),Miguneer(மிகுநீர்),Vehumooththiram(வெகுமூத்திரம்) Innippuneer(இனிப்புநீர). The different classifications of Madhumega noi which have been documented based on the observations of the complaints of the patients in different school of thoughts such as YugiVaithiya Cinthamani-800, Agathiyar

kanmakandam, Theraiyar vaagadam and Thirumoolar Vaithiyam-600 and the complications due to madhumega noi are explained under Madhumega avathaigal.

2.1.2 GUNAPADAM ASPECT

Kadukkai

Botanical name : **Terminalia chebula**

Synonyms : Anthan, Abhayan, Amudham, Devi, Divya, Rohini, Ammai, Abaranam, Aritaki, Varikkai, Jeevandhi Vernacular Names

Tamil	: Kadukkai	English	: Myrobalan
Hindi	: Harre, Harad, Harar	Sanskrit	: Haritaki, Abhaya, Siva, Pathya
Telugu	: Karaka, Karakkaya	Kannadam	: Alalekai

Other Varieties : Visayan, Arokini, Prithivi, Amrita, Sivanthi, Thiruvirti, Abayan Part used : Dried fruit

Properties

Suvai (Taste) : Thuvarppu, Inippu, Kaarppu, Kaippu, Pulippu

Thanmai : (Nature) Veppam

Pirivu (Bio- Transformation): Kaarppu

Actions.

1. Digestive
2. Expectorant
3. Laxative
4. Appetizer
5. Nutrient
6. Hypoglycemic
7. Hypolipidemic

Ingredients and Medicinal uses of Kadukkai Chooranam

Table 2.1 Ingredients and Medicinal uses of Kadukkai Chooranam *(Gunapadam Mooligai Muthal Pagam Page No.201)*

TAMIL NAME	Pharmacological ACTIONS	THERAPEUTIC USES IN SIDDHA
Kadukkai	Anti diabetic, Neuropathy Anti oxidant, Analgesic, Hypolipidemic activity	Mega disorders, Burning sensation of the upper and Lower limb, Thirst, Liver disease, Cardiac disease, General Debility polyurea.

Table 2;2 *Kadukkai vallarien tani manbu* page no 09

TAMIL NAME	Pharmacological ACTIONS	THERAPEUTIC USES IN SIDDHA
Kadukkai	Anti dia betic, Anti oxidant, Analgesic, Hypolipidemic activity	Mega disorders, Burning sensation of the upper and Lower limb, Thirst, Liver disease, Cardiac disease, General Debility polyurea.

Table2;3-*Pathartha guna Sinthamani*, Page no;201

TAMIL NAME	ACTIONS	THERAPEUTIC USES IN SIDDHA
Kadukkai	Anti diabetic, Laxative,Periph eral neuropathy Analgesic,	Mega disorders, Burning sensation of the upper and Lower limb, Thirst, Liver disease, Cardiac disease, Malachikkal

2.2; SIDDHA ASPECT –

Akkini selathumam

These following lines denotes the primary main clinical symptoms of Akkini selathumam is cold and worm extremities,Polyphagia Polydepsia,Burning sensation of the upper and lower limb and generalised weakness.

அக்கினி சிலேட்பம்

என்றதோ ரிருமலொடு கோழை யுண்டா
மெழிலுடம்பு அக்கினியா யெரிய பறந்து
கன்றனவே கால்கையு மழற்சி யாகிக்
கண்ணோடு மூக்குமே யெரிவாய்க் காணுந்
தன்றெனவே தாகமது மிகவுண் டாகுஞ்
சயித்தியமாய்க் கால்கைகள் குளிர்ந்து காணும்
உன்றெனவே யுண்ட பின்பு பசியே யாகு
முறுதியக் கினி சிலேட்மத் துண்மை தானே

\- யூகி வைத்திய சிந்தாமணி-410

இது இருமலில் கோழை விழுதல் ,சர்வாங்கத்திலும் எரிச்சல் ,கைகால்களில் சீதளம்,அதிக தீபனம் என்னும் இக்குணங்களை உண்டாக்கும்.

According to chichitcha rathina deepam ,part 2,Vaithitya chinthamani text book also mentioned the same etiological symptoms of madhumega noi.

பித்த சிலேத்தும தொந்த ரோகங்கள

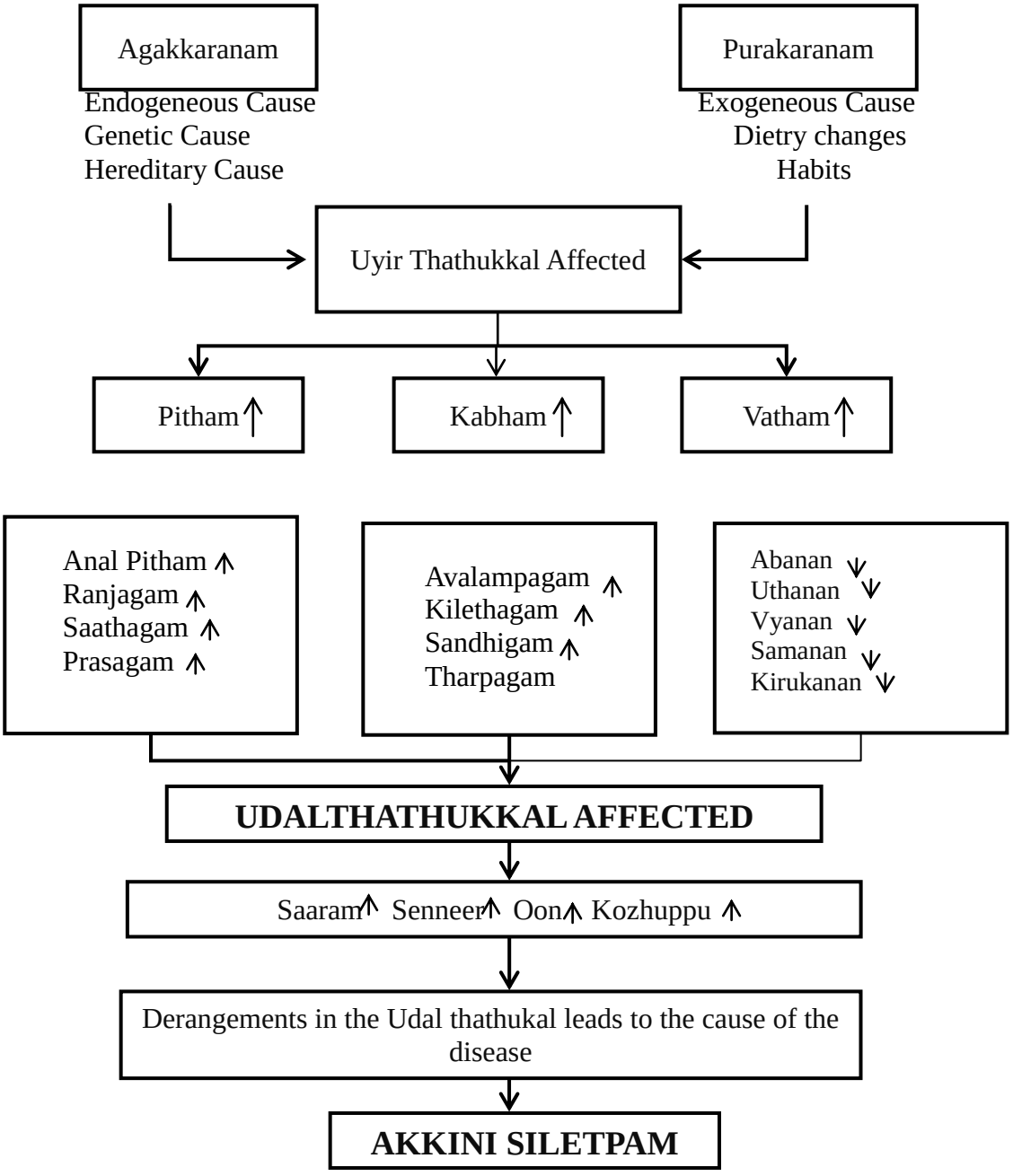
In chichitcha rathina deepam book is mentioned combained pitham and Silothuman can produced Diarrohea,Respiratory disease ,Jaundice, Hematological disease and Epistaxis. These are the complications to produce un treated madhumega noi.

பண்பான பித்தத்தில் சிலேத்துமங் கூடிப்
பரிசித்தால் அதிசுர மிளைப்பே யீளை
கண்காது நயனமலம் தீரும் மஞ்சள்
கனவயிறு பொருமல்மஞ்சள் நோய்கண் ணோவு
உண்போது மறுத்தல்ரத்த விப்புருதி தானு
முளைமாந்தை பீனிசமு ரத்த வீக்கம்
நண்பான காமாலை சோகை வெப்பு
நணுகிவந்த பலபிணியும் நண்ணுந் தானே.

-சிகிச்சாரத்ந தீபம்

2.2.1 ETIO PATHOGENESIS OF THE DISEASE:

Diagramatic alogorhytham presentation in Akkini selathumam. Fig:2;3



2.3. SIDDHA ASPECT OF MADHUMEGAM

Human beings are characterised by the reasoning ability and their striving towards a greater understanding and control over their bodies in relation to the environment. The Thirumoolar in Thirumanthiram,the perception is four conditions were prescribed, they are,

"மறுப்பது உடல்நோய் மருந்தென லாகும்:
மறுப்பது உளநோய் மருந்தென லாகும்:
மறுப்பது நோயை வராதிருக்க,
மறுப்பது சாவை மருந்தென லாகும்"

- Body ailment should be cured
- Mental depression should be annihilated
- Prevention is better than cure
- Man should live a long, healthy life, almost defying death

According to Siddha system, the primordial five elements are Mann (earth), Neer (Water), Thee (fire), Vayu (air) and Aakayam (space), each substance have contained in different proportions. The five element theory of Siddha emphasizes that the forces that are present in Macrocosm (Universe) and are identical with the Microcosm (Human body). Thus a living human is made of these five elements in different combination and the physiological function in the body is mediated by three humours that are made of these five elements. The conglomeration of three humours (Vatham, Pitham, Kapham), Seven basic tissues (Saaram, Senneer, Oon, Kozhuppu, Enpu, Moolai, and Sukkilam/suronitham) and five basic elements (Mann, Neer,Thee, Vazhi and vezhi) is responsible for different structures and functions of human body.

2.3.1.DEFINITION OF MADHUMEGAM

Madhumegam is a disease characterized by frequent of passing urine (polyuria), presence of honey odour in urine on heating. It ultimately deteriorates all the seven Udal thathus (seven fundamental tissues of the body).It was mentioned Guru nadi and Vaithiya chinthamani 800 in following lines,

"இனிப்பான இனிப்பல்ல ஈ வந்தாடும்
ஒரு துளிவாய் விட்டார்கைப் பிணியாய் தோன்றும்"

- குருநாடி

"அண்மையாயடிக் கடிக்கு நீரிறங்கு
மடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையான தடியதனிற்றான் பிடிக்கும்
மிக்கான சடம் வெளுத்து மேனி கன்றும்"

- யூகி வைத்திய சிந்தாமணி-800

2.3.2.ETIOLOGY

The authentic etiological factors described by various siddhars are as follows,

i. Excessive sexual desire.
ii. Excessive intake of high fat or high tryglyceride foods.
iii. Chronic alcoholism.
iv. Obesity
v. Physical inactivity
vi. Psychosomatic stress
vii. Genetic factors are lead to madhumegam.

"கோதையர் கலவி போதை
கொழுத்த மீன் இறைச்சி போதை
பாதுவாய் நெய்யும் பாலும்
பரிவுடன் உண்பீ ராகில்
சோத பாண்டுருவ மிக்க
சுக்கில பிரமேகந்தான்
ஒது நீரிழிவு சேர
வுண்டென அறிந்து கொள்ளே"

-அகத்தியர்-1200

”உற்பவிக்கும் பால் நெய்யால் இறைச்சி கள்ளால்
வுரிசையாய் மீன்தன்னாய் அருவிருந்த
மற்பவிக்கும் பதார்த்தத்தால் மதுரவஸ்தால்
மந்தங்கள் தனைபுசித்தல் வேகாப் பண்டங்
குற்பவிக்கும் குளுத்த வன்ன மங்கை கோஷ்டி
குறித்த நித்திரை தவிர்தல் அக்கினி மந்தம்
தற்பவிக்குந் சரீரந்தான் மிகப்பருத்தற்
சஞ்சலந்தான் மிகப்பயத்தால் தரிக்கும் நோயே”

- யூகி வைத்திய சிந்தாமணி-800

Yugimuni in Yugimuni Vaithiya Kaaviyam is comprehensively described the causes of akkini selathumam in his poem as,

“கட்டளைமிகுந்திட்டாலுங் காலங்கள் தப்பினாலும்
இட்டமாம் பாலும் நெய்யும் ரத்தமும்புளிப்பும் மிஞ்சில்
வட்டமார் முலையார் தங்கள் மயக்கத்தின் கலவியாலும்
நெட்டிலைகோரை போலே நீரிழிவாகுந்தானே”

-யூகிமுனி வைத்திய காவியம்

- **பாலும் நெய்யும் ரத்தமும்புளிப்பும் மிஞ்சில் -Excessive consumption of high fat contents including dairy products**

The food contains high fat diet like ghee, milk and animal flesh are precipitate the cause for obesity, it wis difficult to utilized in insulin level in peripheral tissues (HYPER INSULINEMIA), for produced Type II diabetes.

- **கலவியாலும் -Excessive sexual activities or pervertated sexual behaviour**

According to siddha system of medicine, human body is made up of seven udal thathus. They are saaram (chyle), seneer (blood), oon (bone), kozhupu (cholesterol), enbu (bone), moolai(bonemarrow), sukilam/suronitham (semen/ovum), where each one is derived from the other in the order and the semen/ova being derived from the other six thathus, when wasted injudiciously, results in the deterioration of seven body tissues and gives way to weak immune system and eventually to Madhumegam. More sexual activity can interference in sexual steroid hormone, steroid hormone is resist to produce insulin resistant diabetes. The same was mentioned in Nadi nool,Theran maruthuva bharatham and Guru nadi nool.

“கன்னி மயக்கத்தால் கண்டிடு மேகமே”

- நாடி நூல்

"கிரந்தி புண்ணீரன மேக கீசகனெனுந் துன்மாக்கன்
அருந்ததி என்னும் பாஞ்சாலி யன்னையை கண்ணுற்றானே"

-தேரன் மருத்துவபாரதம்

"ஸ்திரிபோகம் செய்ததினால் வேவு கொண்டு
சிரசுமட்டும் வெந்துருகி கனலே மீறிக்
குறியுடனே மேகந்தான் கொடுமை செய்து
குறைந்து வரும் தாதுவெல்லாம் குன்றிப் போகும்"

-குருநாடி

- **சஞ்சலந்தான் மிகப்பயத்தால் - Psychosomatic Stress**

As per Yugi vaithiya chinthamani-800 was mention in his poem,Stress is produced stress related hormone (5-HT,Stertonin,ACTH..etc),it was produced Oxidative stress and hepatic dysfunction.

"மதங்கொண்டு பெரியோரை வைகையாலும்
மாதர் கற்புநிலைமை தன்னை அழிக்கையாலும்
பதங்கொண்ட சிவயோகி சாபத்தாலும்
பத்துவகை சிலேற்பனங்கள் மேகநீராம்"

- யூகி வைத்திய சிந்தாமணி-800

- **கர்ப்பத்தில் உதிக்கும் நோய் - Hereditary factors**

In modern text book, one of the classification in gestational diabetes,same to described in Thirumoolar in his Thirumanthiram.

"பேறு இளமை இன்பம் பிணி மூப்பு சாக்காடு
ஆறும் கருவில் அமைந்த படி
முறை கேட்கில் ஒன்பது முயற்சியால் வந்தது
துறை கேட்கிற கருப்பத்திற் றுவங்கிய மேகங்கள்
நரை பூத்த கொங்கையாள் நாயகன் மோகத்தால்
மறைபோற்றுங் கருப்பத்தில் வளர்ந்தது மேகமே"

-திருமூலர்

- **கன்ம வினைகள் - Karmia (Genetic disorders)**

Theraiyar vagadam and Agasthiar kanmakandam was stated in madhumegam occurs in genetic disorders (Type I DM).Type 1 diabetes has a codition called type 2 polyglandular auto immune syndrome (HLA-DR3,HLA-DR4 and HLA- DR 7-ADA 2017).

"ஆமப்பா மனிதர்செய்த கன்மத்தாலே
அரகரா மேகமென்று ராசாவாலே
காமப்பால் வீதததாற் வருங்கன்மத்தாலே
கைக்கடங்கா நோய்கள் வருங்கன்மத்தாலே
போமப்பா மேகம் வந்த காரணந்தான்"

-அகத்தியர் கன்ம காண்டம்

"தானே பூருவ விதியதனாற் சாரும் பிணிகளல்லாமல்
மானோர் விழியார் வேட்கையினால் வருந்தும் பிண்ணும் பசியாலே
தானே பொறுத்து உண்கையினால் தாகந் தன்னால் மிகச்சார்ந்து
தானே கமலம் புண்ணாகிச் செய்யும் பிரமியச் செயல்தானே"

-தேரையர்வாகடம்

According to Sarabenthirar Vaithiya Muraikal Neerizhivu Chikitsai text was elaborately dicuss about the following etiological factors for madhumega noi.

மேகமெனும் நீரிழிவு வரும் விதத்தை
விளம்புகின்றேன் முன்செய்த கன்மந்தன்னாற்

றாகமுடன் மதுரபதார்த்தங்கள் நன்றாய்த்
தான் புசிக்கையாலு சித்தத்தின் மங்கை

போக மதிகையாலு முட்டணந்தான்
போதமமிஞ்சுகை யினாலுந்தயிர்,மோர்,நெய்,பால்

வேகமாய்ப் புசிக்கையினாலுங் கொழுத்திறைச்சி
யென்று முண்கையாலுவர் நீருண்கையாலே.

ஆசையுடன் சிறுவழுதலங்காய் தன்னை
யதிகமாயுண்டாலுங் காலந்தப்பிப்
போசனங்கள் செய்தாலு நடையலைச்சல்

போதையாயிருந்தாலுங்கண் விழிக்கையாலுந்
தேசமதிற் பல நீருண்டிடுகையாலே
சிரந்தனிற் சூடதிகங்கொண்டுடனே ரத்தம்
சோஷித்து வேகமதிகமாய்த் தோன்றித்
தொல்லை செய்யும் நீரிழிவுமிருபதாமே

- சரபேந்திரர் வைத்திய முறைகள் நீரிழிவு சிகிச்சை

2.3.3 நோய் எண் (CLASSIFICATION)

Basically madhumegam is classified according to passing frequency and quantity and quality of urine. According to above criteria Madhumega noi was further classified in to twenty types, namely Vatham, Pitham and Kapham. In this main types,further it was divided to four in vatham, in six pitham and ten in kabha disease.

2.3.4 முற்குறிகுணங்கள் (PRIMARY SYMPTOMS OF MADHUMEGAM)

In Siddha text book mentioned that increased abana vayu was affected in kabalam, decreased blood components and dysfunctioned dhasavayukkal,dysphasia is a main cause for madhumegam noi.

"சரியாக மேகத்தால் அபான வாயு
தான் புகைக்கு மேலேறிக் கபாலச்சூடாம்
பெரிதான மேகத்தால் அத்தி வெந்து
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தசவாயுவால் மந்தங்கொண்டு
பெருந்தீனி மலபந்தம் உதானவாயு
வரிவாகித் தேகமெல்லாம் விடநீராலே
மெய்யழிந்த மேகமென்ற திருபதாச்சே"

- சித்த மருத்துவம

The dhanvanthri in Dhanvanthiri Vaithyam Part-II manuscript is explained same to previous siddha litrature of premonitory symptoms of madhumegam.

"மண்டலந் தன்னிலுள்ள மாதர்க்கும் புருடர்க்குங்
கொண்டதோர் சலக்கழிச்சல் கொள்ளுமுன் காணுநோய்கள்
கண்டிடு முடல் கால் கைகள் கான்றழந் தெரிந்து காந்தி
யுண்டநீர் சுவறிக் காட்டி யுடைந்துநீர் கழியுமென்றே"

- தன்வந்திரி வைத்தியம்

2.3.5 குறிகுணங்கள்(SIGNS AND SYMPTOMS OF MADHUMEGAM)

Yugimuni in his Yugivaidhiya chinthamani-800 has described the following common symptoms and signs of 20 types of madhumegam as followed,

- Frequency of Urination (Polyuria)
- Excessive Thirst (Polyphagia)
- Excessive Appetite (Polydipsia)
- Tiredness
- Fatigue
- Irritability
- Progessive of weight loss
- Blurring of vision
- Recurent UTI and Epidymo-orchitis.
- urine may be cold, slimy to touch, brownish yellow in colour and produces white sediments
- Ants and flies are attracted to the site of voided urine
- Passing urine is heated it gives honey odour in nature

"கூறான மேகமது இருபதுக்கும்
குணந்தனை சிவன்சொல்ல தேவிகேட்க
தாறான தாகமொடு சோக மேகந்
தரியாமல் நீரிழிதல் இருமல் மூச்சு
ஆறானஅருசி சத்தி சித்த பிரமை
அடிக்கடிக்குத் தண்ணீர் தானன்னங் கேட்டல்
ஈறான இடுப்புகள் கடுப்பு காணல்
எலும்பு ழற்றலழற்றலோ டெரிவுண்டாகும்"

"எரிவோடு சரீரமெல்லா மறைபட்டாற் போல்
எழிழுடம்பு நோதல் நித்திரை யில்லாமை
மனது சஞ்சலப்படுதல் காற்று வேண்டல்
மெரிவோடு மேல்மூச்சு மிகவுண்டாதல்
விக்கலொடு மயக்கந்தான் மெத்தக் காணல்
தெரிவோடு தேகமெங்கும் வெளுருண்டதால்
தேகமெத்த வாலோபப்படுதல் காணே"

"தண்மையாய் சலந்தானும் பசுப்பு மஞ்சள்
தானிறங்கும் பீசமும் கோசமுங் கடுக்கும்
அண்மையாயடிக்கடிக்குநீரிறங்கும்
அடிக்கடிக்கு அரைநாழி தனிலே தாணும்

"வெண்மையாய் யடியதனிற்றான் பிடிக்கும்
மிக்கான சடம்வெளுத்து மேனிகன்றும்
பண்மையாய்ப் பஞ்வாண்டதனிற் கொல்லும்
பகிர்கின்ற மதுமேகத்தின் பாங்கு தானே"

- யூகி வைத்திய சிந்தாமணி-800

Agasthiyar in Aayulvagadam also same clinical symptoms and signs has to repeated in his poem.

- Burning sensation on hands, legs, Face-Neuralgia
- Dryness of mouth
- Giddiness,vertigo
- Fatiqueness
- Tremors
- Anorexia
- Profuse Sweating

"முகமே காந்தி நெஞ்சுலர்ந்து முறுத்து
முடலு நடுங்கி நகமே பரிந்து சீர் நெகிழ்ந்து
நஞ்சுண்டவர் போல் தேகம் சோர்ந்து பகலுமிரவு முருக்கியுடல்
பகறுமேனியும் தளர்ந்து மிகவே தாவணமுண்டாகும்"

- அகஸ்தியர் ஆயுள்வாகடம்

2.3.6 Common Sign and Symptoms of Vatha, Pitha and Kapha Megam

Based on Yugi, described 20 sub types of Madhumega noikal. The different clinico-pathological conditions are impairement of specific doshas and saptha dhathukkal.The fractional changes in udal dhathus can produced in diabetic complications (Neerlivin avasthikal). According to yugi vaidhiya chinthamani-800 the following clinical feature are vali,azhal and kabha mega noigal,details are given in Table no1.

Table 2;4. Clinical features of different sub types of madhumegam.

Doshas	Types	Specific Signs	Common Symptoms of Doshas
Vatha Megam			
1.	Achiya megam (nei mana neer)	• Urine contains colour of ghee, stickiness and ghee smell. • Polyuria • Weight loss • Death occurs 7 days after disease appeared.	• Burning sensation of hands, feet and face. • Dryness of mouth • Black discolouration of teeth, tongue and throat. • Difficulty in speech • Giddiness • Excessive Thirst • Excessive Appetite • Ache and pain all over the body
2.	Suththa megam (pasu mana neer)	• Urine likes cow's urine and smell • Polyuria • Weight loss and fatigue • Death occurs 15th day after disease appeared.	
3.	Pramiya megam (oon mana neer)	• Polyuria • Smell like blood • Gives honey odour when burned • Killed in 6 months	
4.	Mangisaravi megam (Elamarik kozhuppu mana neer)	• Urine contains particles of flesh and membrane • Give smell of Billy meat washed water (pink). • Polyuria • Death occurs 3-8 days or 5th month	

Pitha Megam			
1.	Appiya megam (yanai matha neer)	• Simile of such patients is given with adult elephant as regards passes of urine. • Sediment like sea sand if boiled • Killed in 6 months	• Burning sensation in all over the body • Emaciation • Excessive perspiration and bad odour • Urine passes like pus, honey, aloe juice • Burning in urethra, scrotum, liver and stomach
2.	Apiramiya megam (kattralai mana neer)	• Polyuria • Aloe smell • Gives putrid odour when boiled • Killed in 3 years	
3.	Sampirna megam (chunna mana neer)	• Urine is like an alkali (ash) solution, in smell, colour and touch. • Killed in 2 years	
4.	Mathumiya megam (thithippu neer)	• Frequency of micturition • Pain in urethra • Honey smell when boiled • White colour sticky precipitation in bottom • Pallor of the body • Killed in 5 years	
5.	Asaththiya (palingu mananeer)	• Dysuria • Quality of urine is turbid & slimy. It is sticky & threads may be demonstrated like gum. • Killed in 5 years	
6.	Arkka megam (muyatkuruthi neer)	• Frequent and excessive micturition • Urine red in colour like hare's blood and meat smell. • Dysuria • Killed in 9^{th} month	

Kapha Megam			
1.	Vasa megam (vasa neer)	• Urine contains fat (vasa) and smell • Pain in penis and scrotum • Death occurs within 7 years	
2.	Uththama megam (theli neer)	• Clear urine in larger quantity without odour, feels cold sensation while passing urine. • Killed in 10 years	
3.	Machcha megam(moolai neer)	• Urine seems to like contains bone marrow (majjai). • Polyuria • Putrid smell • Life span-5 years	
4.	Akiha megam (ela neer)	• Urine like tender coconut water and smell. • Gives coconut oil smell when boiled • Polyuria • Weight loss • Thirst • Anxiety • Killed in 7 years	• Obesity • Pallor of body • Skin rashes like itching, ulcers and allergic rashes • Excessive Thirst • Excessive Appetite • Cough Sputum collection in throat
5.	Surari megam (kal neer)	• Urine-white in colour and frothy like toddy and smell. • Fatigue • Killed in 7^{th} year	
6.	Sukkila megam (thavala neer)	• Patient passes urine similar to quality of semen or semen itself may be mixed with urine. • Black colour sediment like liver after boiled • Killed in 3 years	

7.	Udhaha megam (kalu neer)	• Urine incontinence present • Precipitation like lime of conch • Body odour present • Killed in a year	
8.	Pinani megam (then neer)	• Enormous urine output like honey and smell • Sediment like wax • Ants and flies are attracted to the site of voided urine • Honey odour present in body • Killed in 5 months	
9.	Lavana megam (uppu neer)	• Urine seems to be salty and white and it's odour. • Polyuria • Alkali ash precipitation • Sediment salt when boiled • Weight loss, worries, loss of appetite, and indigestion • Killed in 15 years	
10.	Thayiththiya megam (eraichchi neer)	• Urine red in colour and smell like meat washed water. • Dysuria • Polyuria • Killed in 3rd year	

வளிக்குற்றத்தால் வரும் மேகநீர் நோய்:

"ஆச்சென்ற நாலும்முங் குணத்தைக் கேளா
யழகான கைகால்கண் ணுடல ழற்றும்
நாச்சென்ற நாவறளும் பல்லு நாக்கு
நடுத்தொண்டை கறுப்பேறு முதலெட் டாந்தான்
பேச்சென்ற நாவறளும் பல்லு நாக்கு
நடுத்தொண்டை கறுப்பேறு முதலெட் டாந்தான்
பேச்சென்ற பிலசஷயமாங் கண்மேல் நோக்கும்

பெருகவன்னந் தண்ணீரு மிகவே வாங்குந்
தாச்சென்ற சரீரந்தான் கத்தி வெட்டுத்
தான்போலக் கடுத்துமே தழலுண்டாமே”

- யூகி வைத்திய சிந்தாமணி-800

அழல் குற்றத்தால் பிறக்கும் மேகநீர் நோய்கள்:

“அறியவே பித்தசலமாறுமே தான்
அங்கமதிற் செய்கின்ற குணத்தைக் கேளாய்
தறியவே உடல்வற்றி எரிவுண்டாகும்
சடத்திலுந்தான் நீரிலுந்தான் கவிச்சுண்டாகும்
தெறியவே சீப்போலுங் கற்றாழை போலுந்
சேல்போலுந் தேன்போலு நாற்றமுண்டாம்
வெறியவே பீசத்திற் கோசத்தில் குத்தல்
மிகுமீரல் நாபியிலும் வேக்காடாமே”

“வேக்காய் விரணமுண்டாய் வாய்தானாலும்
விக்கலோடு அருதியாய்ச் சுரமுண்டாகும்
தீக்காடாய் தேகந்தான் கிடக்கொட்டாது
தியக்கமொடு மூர்ச்சையுண்டா மயக்கமாச்சே
சாக்காடாய் நாவறளுங் கண்ணீர் தாகஞ்
சாத்தியொரு சரீரமெலாந் தளர்ச்சி யாகுந்
தாக்காடாய் மலசலந்தான் மிகவுண்டாகுந்
தாக்காடாய் மலசலந்தான் மிகவுண்டாகுஞ்
சமகுணந்தான் பித்தசல மாறுமாச்சே”

- யூகி வைத்திய சிந்தாமணி-800

ஐயக்குற்றத்தால் பிறக்கும் மேகநீர்நோய்கள்:

“தசமான பத்துக்குங் குணத்தை கேளாய்
சரீரந்தான் பருத்துமே வெளுப்புண்டாகும்
அசமான தினவுண்டா மடிக்கடிக்கு
கசமான விருமலுடன் கோழை யுண்டாங்
கனவரிவா யாயாச முழலையாகுங்
குசமான குணங்களையெல்லாம் சிலேட்டுமந் தன்னில்
கொடிய சலக்குணமென்று கூறினாரே”

- யூகி வைத்திய சிந்தாமணி-800

2.3.7. முக்குற்ற முதலிய வேறுபாடுகள் (PATHOGENESIS)

The direct inference from these poems is that all Siddhars attribute diabetes mainly due to excessive indulgence in sex which results in total loss of body strength as a whole including the nervous system. Due to the intrinsic, extrinsic and other causes tridoshas are mainly affected. Initially the pitha dosham has vitiated and causes burning sensation of the body and altered vayus,further altered Kapham and Vatham and udal kattugal can disturbed normal physiological functions. The severity of the disease is measured by the functions of three doshas and seven udal thathus. Debilitation and other sequence of disease will be occurring due to loss of appetite and loss of body strength.

According to theriyar in vagada nol and pathinen siddhar nadi nool is same to be repeated in below poem.The malnourishment of saptha dhathus is produced polyurea,sweety odour in urine and loss of strength is an important characteristic feature of the Mega neer.

"பகர்பித்த விந்தையலாது மேகம் வராது"

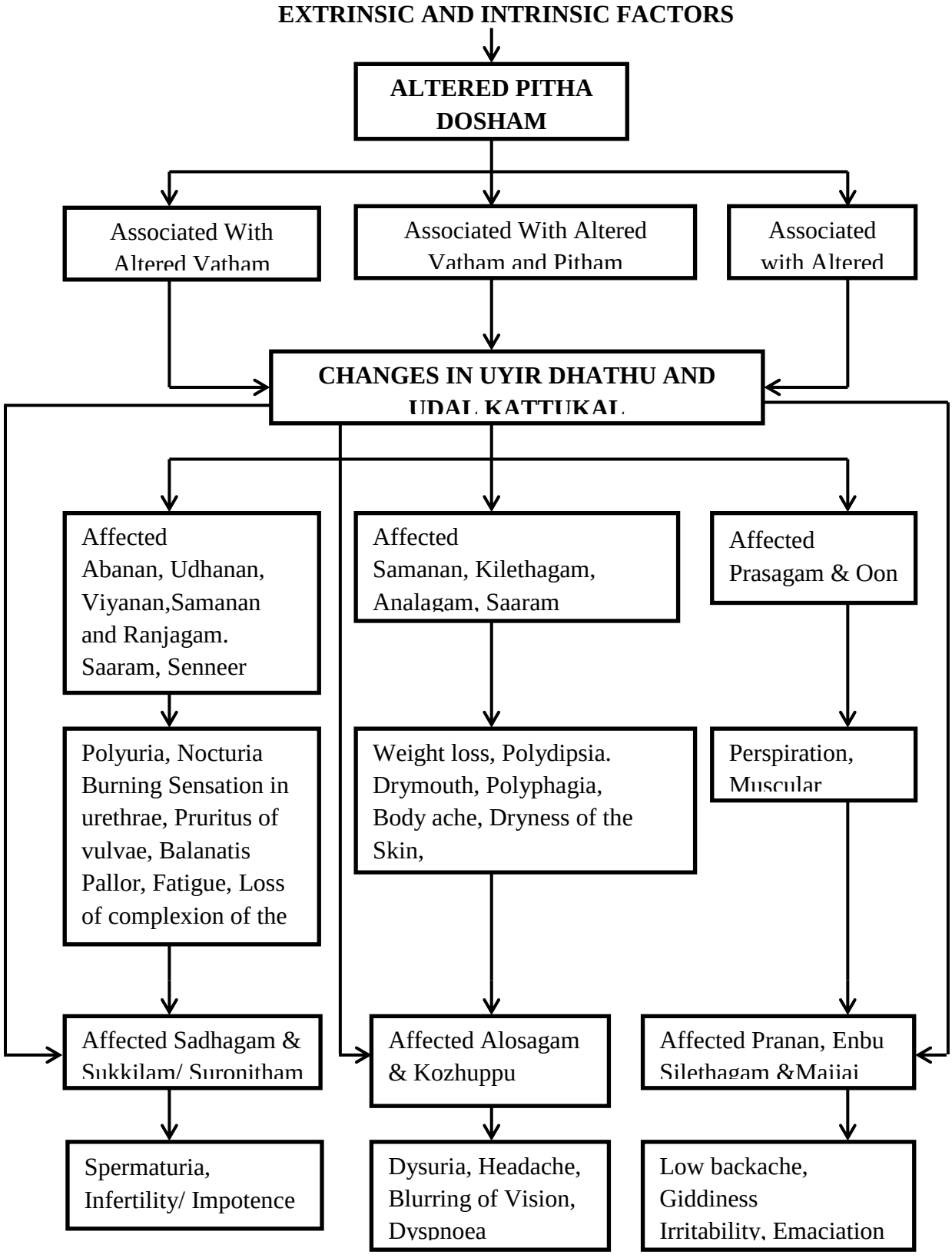
- தேரையர்

"குறியுடனே மேகந்தான்
கொடுமை செய்து
குறைந்துவருந் தாதுவெல்லாங்
குன்றிப்போகும்"

- பதிணென் சித்தர் நாடி நூல்

Diagramatic presentation in pathology for Madhumeganoi.Fig.2;4

PATHOGENESIS OF MADHUMEGAM



2.3.8 மதுமேக நோயில் காணும் பத்துவகை அவத்தைகள் (COMPLICATIONS OF DISEASE)

Saint Yugi well elaborated in the text book of yugi chinthamani, the onset of the following sufferings as avathaigal will be followed gradually if the disease is not controlled or left untreated.

Avathai-1: Progressive weight gain and dilatation of urinary meatus

Avathai-2: Excessive urination, disorder of semen (polyuria, Asthenospermia)

Avathai-3: Dryness of the tongue and gaseous abdominal distension (polydipsia, and diabetic gastro enteropathy)

Avathai-4: Excessive thirst may leads to excessive fluid loss (Encephalopathy, polyphagia, Diabetic metabolic encephalopathy)

Avathai-5: Frequency of urination, spermatorrhoea (chronic renal failure)

Avathai-6: Patient awakening in bed, breathlessness (metabolic syndrome)

Avathai-7: Recurrent nausea with vomiting, breathlessness (metabolic Syndrome)

Avathai-8: Chronic ulcer, abscess or carbuncles are present in body (Diabetic Ulcer)

Avathai-9: Immoral behaviours, watery diarrhoea (Superadded opportunistic infections)

Avathai-10: Pulmonary and extra pulmonary tuberculosis

"காணவே முதலவத்தைச் சரீரந் தானுங்
கனமாகப் பருத்திறுகி நீர்த்து வாரம்
வேணவே வேண்டாக்கி யகலம் பண்ணு
மிக்கவரண் டாமவத்தை விளம்பக் கேளாய்
மூணவே மூத்திரப்பீ டையுமாச் சுக்ல
முகமுழுகித் தேஜசுதான் மிகவே குன்றும்
நாணவே மூன்றாகு மவத்தைக் குத்தான்
நாவறளும் வாயுவது மீறுந் தானே"

"தானான நாலவத்தை யங்க தாகஞ்
சன்னியது பாதமுண்டா மைந் வத்தைத்
தேனான நீர் பெருகுந் தாதுநஷ்டம்

நிலையாற மவத்தையுடற் கிடைகொள்ளாது
மூனான மூர்ச்சைவரு மேழ வத்தை
மிக்கவரோ கசஞ்சுவாசந் தேக சாட்டியம்
ஏனான எட்டாவ தவத்தை தானே
எழுகிரந்தி பிளவை யுந்தான் மிகவுண்டாமே

உண்டாகு மொன்பதா மவத்தைக் கேளாய்
உழக்கான வதிசாரங் கிருமி யுண்டாம்
பண்டான பத்தாந்தா வைத்தை கேளாய்
பாரமாம் சயங்கண்டு பரத்துக் கேகும்"

- யூகி வைத்திய சிந்தாமணி-800

According to the different school of thoughts, the above 3 avathaigal will be cured with medicines and up to nine avathaigal can treat the disease.

2.3.9 தீரும் தீராதவை (PROGNOSIS OF THE DISEASE)

The imbalance in the ratio of Vatham, Pitham and Kapham affects the five vayus (abanan, udhanan, viyanan, samanan, pranan), seven udalkattukal,The prognosis is depentened to Mukkutram and dhasavayukkal.

The second thought of Yugi is added to three categories are good prognosis namely,

1. **Sadhyam (Manageable) -** Kapha megam (10)
2. **Yapyam (Palliative) -** Pitha megam (6)
3. **Asadhyam (Complicated) -** Vatha megam (4)

"செய்யவே வச்சிரமாந் தண்ட மான
செயமான முதுகுத்தண்டைப் பற்றி நிற்கும்
பெய்யவே பெருநரம்பில் மேகந் தானும்
பிறக்குமென்றே தானறிந்து வாதந் தன்னால்
பிய்யவே பிறனந்தசலம் நால சாத்தியம்
பித்தத்திற் பிறந்தசலமாறும் யாப்யம்
கையவே சேட்டுமத்திற் பிறந்த பத்தும்
பரமனுரைத் தார்சாத்தியம் பராப ரிக்கே"

- யூகி வைத்திய சிந்தாமணி-800

The diarrhea, generalised edema and MDR tuberculosis and other respirotary complications, abdominal pain, carbuncle is complications can produce untreatable diabetes,it was mentioned in kannusamiyam book.

"நீர்நோயினிலதிசாரமு
னிமிர்வீக்க மிளைப்பு
மார்மூச்சுறல் விக்கலடிக்
கடியேவரல் வயிற்றில்
சேர்நோயோடு பிளவைவரல்
தீராக்குறி யென்றே
நார்கொண்டுறை செய்தாரிதை
நன்றாயறி வாயே"

- கண்ணுசாமியம்

Accoding to Sathaga Nadi is discussed about increased vatham,vatham is combained with Chronic abdominal discomport and respirotary disease,Weight loss is a poor prognosis of diabetes.

"துதிப்பான மேகத்தில் நீரிழிவு மாகா
தோன்றியநீ ரிழிவுதன்னில் வாதமுமாகா
மதிப்பான வாதத்தில் வயிற்றுளைச்சலாகா
வருமுளைச்சல் தன்னில்வாயு கொழுத்து மாகா
கெதிப்பான வாய்வதிலே விக்க லாகா
கூண்டவிக்கல் தனிலிளைப்பு கொழுத்த லாகா
குதிப்பான இளைப்பதிலே சுவாசம் வந்து
கலந்தாலும் மரணம் என்று கருதலாமே"

- சதக நாடி

2.3.10 நோய் கணிப்பு (DIAGNOSIS OF THE DISEASE)

"நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்"

- தேரையா

In Siddha System of Medicine Eight different parameters of diagnosis have been devised to establish the exact underlying pathology known as envagai

thervugal,namely Nadi, Sparisam, Na, Niram, Mozhi, Vizhi, Malamand Moothiram and confirmation by the history,clinical symptoms and lab parameters.

Nadi Nadai paritchai (Reading of Pulse)

"பார்த்திடு மூன்றும் பதிந்து மெலிந்து நிற்கில்
தேர்ந்திடு மேகம் வந்தோன்றியே பொருந்தி மெய்யில்"

- திருமூலர் நாடி

- The most important parameter of diagnosis is Nadi.
- The three Vatha, Pitha and Kapha naadis are feeble, the corresponding derangement in the doshas leads to Madhumegam.

The second opinion of Thirumoolar,Pitha and vatha variation of clinical symptoms as followed, excessive hunger, thirst, emaciation and passing of large quantities of urine with sweet taste.

"இருமிய பித்தமும் வாதமும் கூடில்
மருவுல மேகம் வாருதி போலாடும்
உருவம் வேறொரு முண்டவுடற் காய்ந்திடும்
உருகவே வூனோடு உறிஞ்சி இனிக்குமே"

- திருமூலர்நாடி

Thirumoolar state, Vatham and kapham is combined together,this was mentioned in thirumoolar nadi,further appearance of Kudila Nadi is movement of worm and Paripoorana nadi are quoted that,in the developed stage of the disease the vatha, pitha and kapha nadi will be feeble.

"இனிக்கின்ற வாதத்திடை சேரில் ஐயந்தான்
பனிக்கின்ற கள்ளுப் பதனிபோல் நீரோடும்
கனிக்கின்று மேனி கரைந்து வெளுப்பேறும்
கனிக்குமது மேகந் தப்போதையமே"

- திருமூலர்நாடி.

"பார்த்திடு மூன்றும் பதிந்து மெலிந்து நிற்கில்
தேர்ந்திடு மேகம் வந்தோன்றியே பொருந்தி மெய்யில்"

- திருமூலர் நாடி

"துரணமுடன் நீர்ப்பாடு கெர்ப்பப் பாடாணாற்
சொல்லுகிறேன் நாடியெல்லாந் கழன்று காணும்"

- பரிபூரணநாடி

"நீர்மேகமானவர்க்கு நாடி தானும்
நீர்மயமாய் நாடியெல்லாம் பலமே கெட்டுக்
கார்மேகம் போலேவந் தெரிமேல் புரண்டு
விழும்புழுப் போலவே புரண்டு காட்டும்"

- பரிபூரணநாடி

The increased Pitha naadi is produced an excessive burning sensation,this willbe poor prognosis of diabetes.

"பற்பிடிக்க மேகம் என்றால் பித்தமீறும்
பாலகனே காங்கை கொண்டு நீராம் பாரே"

- பரிபூரணநாடி

- SPARISAM (SENSATION OF PATIENT DURING TOUCH)

Warm (or)Dry, pricking pain (or)numbness all over the body. In mega neer due to Vatha, Pitha and Kapha dosham, have cold sensation in extremities.

- **NA (EXAMINATION OF TONGUE)**

In tongue examination following things are should be consider as follows,

- ❖ Niram (colour) - Pale in kapha neer
  Black in vathaneer
  Yellow pitha neer
- ❖ Thanmai (character) - Dry and fissured
- Pulan (sense) - Lethergic and Fatiqueness
- ❖ Umizh neer (salivary secretion) - Saliva in sweet taste

- **NIRAM (EXAMINATION OF COLOUR AND COMPLEXION)**

It is differ from their original complexion on the skin. In madhumega disease pale or dark complexion is common.

- **MOZHI (EXAMINATION OF SPEECH)**

Speech due to increase of pitham, the patient is likely to suffer from tiredness and giddinees, therefore the bound of speech become low pitched voice.

- **VIZHI (EXAMINATION OF EYE)**

In madhumegam visual disturbances is common (blurred of vision, glaucoma and pre mature cataract) may be present and following things also should consider,

- Niram (colour) -red/pale
- Thanmai (character) -dry
- Pulan (sense) -reduced touch sensation impairment in vision

- **MALAM (EXAMINATION OF STOOL)**

- Niram (colour)
- Nurai (frothy)
- Elagal/ erugal (consistency) are should be consider in examination of stools.When Vatham is in high proportion there is constipation, with increase of pitham there exists diarrhoea and increase in kapham results in white, milky motion.

- **MOOTHIRAM (EXAMINATION OF URINE)**

Urine examination is done under two categories,

a) Neerkuri (The common nature of urine)
b) Neikuri (oil drop method)

a) Neerkuri (The Common Nature of Urine):

The following points have to be taken into account in the urine examination:

- Colour
- Weight and density
- Odour
- Froth
- Quantity

"வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென்
றைந்தியலுளவவை யறைகுது முறையே"

The common features of neerkuri are,

- Niram (colour) - crystal clear urine
- Weight and density - thickening of the urine
- Manam (odour) - honey smell
- Nurai (froth) - increased
- Enjal (deposits) - small deposits in urine

Theriyar in neerkuri and nei kuri script is explained,urine is white (crystal)in colour, it indicates of kapha neer and poor prognosis.

"வெண்மையுற்று மிகத் தெளிவுடைத்தேல்
உண்மையாந் சுத்த சீதளத் துதகமார்
இந்நீர்ப் பசப்படாதித்வனுடைய யந்தரம்
முந்நீர் பெருக்கமழிவான் உய்தவொக்குமே"

- தேரையர் நீர்க்குறி நெய்க்குறி

b) Neikuri (Oil Drop) Method:

A drop of gingely oil is dropped in to a wide vessel containing the urine to be tested and kept in sunlight. The variations of three doshas diagnosed by shape and spreading of gingely oil on the surface of urine.

"குறியதுகேளும் நீரில் குறைத்தலை போலுந் தோன்றில்
பிறிந்திடுமுடலைபோலும் பெருங் கமண்டலம்போல்தானும்
வறிந்திடச்சாதியம்மீதாம் வலிபில மனுவோர்க்கென்று
செறிந்திடுமுனிவர்தாமுஞ் செப்பியகுறிப்பதாமே."

-யூகிமுனி வைத்திய காவியம்

According to yuki muni vaithiya kavium.,the apperance of spreading oil in the shape of skull and human body is indicated the good prognosis of diabetes.The irregular round shape is indicated for poor prognosis of diabetes.

"கையினிலெண்ணைவாங்கி கழிந்த நீர்தன்னிற்குத்த
செய்ததுவட்டமாகுஞ் சேருந்தோரணம்போல்தானும்
ஐயமுமில்லைகண்டாய் சாத்தியமல்லவென்று
துய்யநன்முனிவர்தானுஞ் சொல்லியகுறிப்பதாமே"

-யூகிமுனி வைத்திய காவியம்

Vatha Neer:

"மூளையும் நிணமும்போல முறிந்த சுக்கிலமே போல
ஆளரி வேலுங்கோலு மணங்கனும் அம்புபோல
நீளிய நரம்புபோல நீர்தனி லெண்ணெய் காணில்
வாளினை வென்ற கண்ணாய்!வாதத்தின் கூறுதானே"

- தன்வந்திரி வைத்தியம

According to Thanvanthiri Vaithiyam, Vatha madhumega noi is an appearance of sperm,brain and unusal urine smell indicated for vatha noi

Pitha Neer:

"பையரவல்குல் மாதே! பாருள் ளோர்கிழிந்த நீரிற்
கையிலேயெண்ணெ வாங்கிக் கழிந்ததோர் துரும்பாற்குத்த
மையறுமேனியொத்த வட்டஞ் செய்திருக்குமாகில்
அய்யுறு நீரதாகு மல்லதாற் பித்தமாமே"

- தன்வந்திரி வைத்தியம்

The urine is dark in colour.they indicated pitha type of madhumega noi.

In Siddha Maruthuva Noi Thoguthi- part -I, Mega roga Nithanam is well defined as the prognosis of madhumega disease,

- The oil drop doesn't spread and placed like circle of eye and then adhere to the mouth of the dish is indicate the disease curable.
- When the oil drop sink into the bottom and given sudden spread then oil and urine mixed is known as incurable.
- The oil is spreading quickely,disappeared in early,oil easily to mixed with urine is poor prognosis of diabetes.It was explained in below lines.

"பாராய் நீர் பாண்டத்தில் பாங்காயதின்மேல் நல்லெண்ணெய்
சீராய் ஒரு துளி விட்டாக்கால் சிதறி ஓடி போகாமல்
நேராய் நின்று கண் வட்டம் போல் நெருங்கி சட்டிவாய் எங்கும்
சேர நெருங்கி நிர்க்கிலுமே தீரும் இதற்கு மருந்து செய்யே

செய்யும் வகையது கேளாய் சிறந்த நீரதிலே எண்ணெயிட்டால்
பைய்ய கீழே தாழ்ந்திடினும் பதறி ஓடி சிதறிடிலும்
நெய்யும் நீரும் ஒன்றாக மிகவே கூடி கலந்திடினும்
உய்யும் வகையது தீராது உத்தமம் எல்லாம் மத்திபமே

கையினால் எண்ணெய் வாங்கி கழிந்த நீர் தன்னில் ஊற்றி
ஒய்யுறில் ஐயமாகும் ஒழிந்திடில் பித்தமாகும்
மெய்யுறும் எண்ணெய் தாளில் மிகுந்ததோர் வாதமாகும்
பொய்யல்ல இம் மூன்றுக்கும் புத்தியாய் அறிந்து பாரே"

-மேகரோக நிதானம்-சித்தமருத்துவ தொகுதி

2.3.11 நோய்க்கணிப்பு விவாதம் (DIFFERENTIAL DIAGNOSIS)

- Theli Neer (Diabetes insipidus)
- Neer Kiricharam (Urinary Tract Infection)

2.3.12 மருத்துவம் (LINE OF TREATMENT)

In Siddha system of medicine is not only for treat the disease, but also prevent the disease and improve the immunity. It is mentioned in text book Thirumoolar-800

Kaapu	-	Prevention
Neekkam	-	Treatment
Niraivu	-	Restoration

"வைத்திய செயல் வைத்தியமாமே
பலவாறு மாறுதலடைந்து கெடுக்கின்ற உடலை நிலைக்கும்படி
மாறுதல் அணுகாமலும் ஒரே தன்மையாக
செய்தும் அதனாலாஞ் செயிலக் குறைவின்றி
நடக்கச் செய்வது தெதுவோ அதுவே வைத்தியம்"

-திருமூலர்-800

Over intake or consuming unbalanced incompatible diet is considered as the prime causative factor to disturb the tridosha equilibrium leading to multitude of ailments. Special attention has been rendered to the diet regimen in madhumegam which is evident in Agasthiyar Kanma Kandam, Yugi Vaithiya Cinthamani-800 and Agasthiyar Ayurvedam-1200.

Since the pathogenesis of madhumegam is mainly depends on the altered doshas,

- Restoring the chemical constitution of a dosha
- Sub-due their levels if they are excited
- Stimulate their levels if they are subdued.

Observing the precautions and following the directions given by a physician on the three Ds (Drug, Diet and Deeds) of health, a person can achieve the state of equilibrium.The aim of treatment is normalise the altered tridoshas and affected vayus and seven udalkattugal. Moreover, as the Akkini selathumam disease is caused by many other causes, it should be treated for primary causes.

"வேர்பாரு தழைபாரு மிஞ்சினக்கால் மெல்லமெல்ல
பற்ப செந்தூரம் பாரே"

The basic treatment of Siddha, neutrilize the three doshas and first line of treatment is plant orgin and second line of treatment is tobe use in metals and minerals preparation.

The following basic principles are added to prescribed medicine.The selection of drug is classified in three varieties, namely

- ஒப்புரை
- எதிருரை
- கலப்புரை

The Siddha literature reveals that, the following types of diet regimens with or without internal treatments.

PATHIYA - APATHIYAM (DIET-DOS & DON'TS)

The diet should be strengthen the udalkattugal and control the urine output. All the below points are mentioned, to be avoid more carbohydrate foods, low glycemic contents fruits, more green leaves and vegatables are adviced in diabetic patients.

A) **ADD:**

- Rice Or Kanji -Clear Soups, Hand Pounded Boiled Rice, Mani Samba Rice, Kezhvaragu, Kambu, Thinai
- Unripe Vegetables -Pahal, Surai, Vendai, Kathari, Avarai, Murungai, Vellai Mullangi, China Venkayam, Atti, Kovai
- Greens -Neerarai Keerai, Kothamalli, Puthina, Karivepilai, Kavaipoo, Puliyaarai, Murungai, Vasalai, Ponmusuttai, Vallarai, Manathakkali, Surai, Kadivasalai, Keerai Thandu
- Fruits -Koiya, Mathulai, Naval, Nelli
- Pulses -Ulunthu, Pasipayaru
- Dairy Products -Cow's Butter Milk
- Non-Vegetarian Diet-Ayirai Meen (Loach)

B) AVOID:

The following foods and milk products are avoid during the line treatment,

- Sweets, backery, products such as cakes, pastries, cream biscuits, concentrated milk preparations
- Ice-creams, soft drinks, fruit juices
- Tubers like potato, sweet potato, yam, carrot, beet root and colacasia.
- Saturated fats ghee and butter
- Nuts, dry fruits like pista, badam, cashew
- Fatty meats, organ meat such as liver, kidney, brain, heart, egg yolk may be avoided.
- **YOGAM:**

The following yogasanam are adviced in diabetes patients,

- Pranayamam
- Sarvaangasanam
- Halasanam
- Patchimothasanam
- Thanurasanam
- Shavasanam
- Salapasanam
- Mayurasanam
- pujangasanam
- Padmasanam

PREVENTION

- Sprouted granules, ginger added with food
- Regular walking (6km/hr)
- Avoid tabacco/ alcohol
- Excessive sexual activity
- Take sprouted fenugreek,onian, garlic, turmeric, terminalia chebula, Indian goosbery

2.3. MODERN ASPECT - DIABETES MELLUTUS

2.3.1 Definition and description of diabetes mellitus

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels. Several pathogenic processes are involved in the development of diabetes.

These range from autoimmune destruction of the pancreatic β-cells with consequent insulin deficiency to abnormalities that result in resistance to insulin action. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in diabetes is deficient action of insulin on target tissues. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulinatone or more points in the complex path ways of hormone action.Impairmen to insulin secretion and defects in insulin action frequently coexistin the same patient, and it isoftenunclear which abnormality, if either alone, is the primary cause of the hyperglycemia. The Symptoms of marked hyperglycemia include polyuria, polydipsia, weight loss, sometimes with polyphagia, and blurred vision, Impairment of growth and susceptibility to certain infections may also accompany chronic hyperglycemia. Acute, life-threatening consequences of uncontrolled diabetes are hyperglycemia with ketoacidosis or the nonketotic hyperosmolar syndrome.

Long-term complications of diabetes include retinopathy with potential loss of vision; nephropathy leading to renal failure, peripheral neuropathy with risk of foot ulcers, amputations, and Charcot joints; and autonomic neuropathy causing gastro intestinal, genitor urinary, and cardiovascular symptoms and sexual dysfunction. Patients with diabetes have an increased incidence of atherosclerotic cardiovascular, peripheral arterial and cerebrovascular disease. Hypertension and abnormalities of lipoprotein metabolism are often found in people with diabetes(Rodica Pop-Busui.et al.2007)

2.3.2Epidemiology

The mortality rate of diabetes mellitus is high, and is 5^{th} rank in the ten major causes of death in southern part of India. The rising prevelance of diabetes is associated with industrialization and socioeconomic development. Although the prevelance of type-I and II diabetes mellitus is increasing world wide. The prevelance of type-II diabetes mellitus is expected increase more rapidly in future because of increasing obesity and reduced physical activity. The WHO estimates that 75 percent of the 300 million adults with diabetes in 2025 will live in devolopig countries.

2.3.3 Classification of diabetes mellitus

Etiologic classification of diabetes mellitus:

I. Type-1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic

II. Type-2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)

III. Other specific types
   A. Genetic defects of β-cell function
      1. Chromosome 12, HNF-1α (MODY3)
      2. Chromosome 7, glucokinase (MODY2)
      3. Chromosome 20, HNF-4α (MODY1)
      4. Chromosome 13, insulin promoter factor-1 (IPF-1; MODY4)
      5. Chromosome 17, HNF-1β (MODY5)
      6. Chromosome 2, *NeuroD1* (MODY6)
      7. Mitochondrial DNA
      8. Others

B. Genetic defects in insulin action
    1. Type A insulin resistance
    2. Leprechaunism
    3. Rabson-Mendenhall syndrome
    4. Lipoatrophic diabetes
    5. Others
C. Diseases of the exocrine pancreas
    1. Pancreatitis
    2. Trauma/pancreatectomy
    3. Neoplasia
    4. Cystic fibrosis
    5. Hemochromatosis
    6. Fibrocalculous pancreatopathy
    7. Others
D. Endocrinopathies
    1. Acromegaly
    2. Cushing's syndrome
    3. Glucagonoma
    4. Pheochromocytoma
    5. Hyperthyroidism
    6. Somatostatinoma
    7. Aldosteronoma
    8. Others
E. Drug or chemical induced
    1. Vacor
    2. Pentamidine
    3. Nicotinic acid
    4. Glucocorticoids
    5. Thyroid hormone
    6. Diazoxide
    7. β-adrenergic agonists
    8. Thiazides
    9. Dilantin

10. γ-Interferon
11. Others

F. Infections
    1. Congenital rubella
    2. Cytomegalovirus
    3. Others

G. Uncommon forms of immune-mediated diabetes
    1. "Stiff-man" syndrome
    2. Anti-insulin receptor antibodies
    3. Others

H. Other genetic syndromes sometimes associated with diabetes
    1. Down syndrome
    2. Klinefelter syndrome
    3. Turner syndrome
    4. Wolfram syndrome
    5. Friedreich ataxia
    6. Huntington chorea
    7. Laurence-Moon-Biedl syndrome
    8. Myotonic dystrophy
    9. Porphyria
    10. Prader-Willi syndrome
    11. Others

IV. Gestational diabetes mellitus

Type-I diabetes characterized by deficiency of insulin due to destructive lesions of pancreatic β-cells; usually progresses to the stage of absolute insulin deficiency. Typically, it occurs in young people with acute-onset with typical symptoms of diabetes together with weight loss and tendency to ketosis, but type 1diabetes may occur at any age, sometimes with slow progression.

Type-II diabetes is caused by a combination of decreased insulin secretion and decreased insulin sensitivity. Typically, the early stage of type-II diabetes is characterized by insulin resistance and decreased ability for insulin secretion causing excessive post-prandial hyperglycaemia. This is followed by a gradually deteriorating first-phase insulin response to increased blood glucose concentrations.Type-II

diabetes, comprising over 90% of adults with diabetes, typically develops after middle age. The patients are often obese or have been obese in the past and have typically been physically inactive. Ketoacidosis is uncommon, but may occur in the presence of severe infection or severe stress. Diabetes related to specific single genetic mutations that may lead to rare forms of diabetes, as for instance Maturity Onset Diabetes of the Young (MODY).Diabetes secondary to other pathological conditions or diseases (as a result of pancreatitis, trauma, or surgery of pancreas).Drug or chemically induced diabetes.

TABLE

CRITERIA FOR DIABETIC DIAGNOSIS. FIG. 2:5

ADA Diagnostic Criteria: Normal, Diabetes, and Pre-diabetes

Clinical Practice Recommendations 2010

	Parameter	Normal	Diabetes	Pre-diabetes	Method
1	Fasting Plasma Glucose (mg/dl)	<100	≥126	100–125	No caloric intake for at least 8 h
2	2–h plasma glucose on OGTT (mg/dl)	<140	≥200	140–199	WHO method: 75 g glucose load
3	Random plasma glucose (mg/dl)	<140	≥200	-	with classic symptoms of hyperglycemia or crisis
4	A1C %	<5.7	≥6.5	5.7 – 6.4	NGSP certified method standardized to the DCCT assay

In the absence of unequivocal hyperglycemia, criteria 1, 2, and 4 should be confirmed by repeat testing

The clinical classification also comprises different stages of hyperglycaemia, reflecting the natural history of absolute or relative insulin deficiency progressing from normoglycaemia to diabetes. The following criteria for diagnosis of diabetes (American Diabetic Association.2010).

2.3.4 Complications of diabetic mellitus

2.3.4.1 Acute Complications

The usual clinical symptoms of DM include polyuria, polydipsia, weight loss, fatigue, weakness, blurred vision, frequent superficial infections, and poor wound

healing. However, patients can occasionally present with acute complications, such as hypoglycaemia, diabetic ketoacidosis, hyperosmolar non-ketotic coma.

2.3.4.2 Chronic Complications

The major chronic complications of DM are usually microvascular, neuropathic and macrovascular in nature. The microvascular and neuropathic complications present as retinopathy, nephropathy, peripheral neuropathy, autonomic neuropathy and foot disease. The macrovascular complications present as myocardial infarction/ ischaemia, transient ischaemic attack, stroke and claudicationhave also indicated that other chronic complications of diabetes may be non vascular, e.g. gastroparesis, infections and skin changes.

2.3.4.3 Macrovascular Complications

Almdal et al 2004 was mentioned, indicated that type-II diabetes typically occurs in the setting of the metabolic syndrome, which also includes abdominal obesity, hypertension, hyperlipidaemia, and increased coagulability. These other factors can also act to promote cardiovascular disease. In this setting of multiple risk factors, type-II diabetes itself acts as an independent risk factor for the development of ischemic disease, stroke, and death. Diabetic retinopathy which occurs in all forms of diabetes is the commonest cause of blindness in adults in most developed countries. The development of retinopathy, as with all diabetic complications, depends on the duration of the disease. The clinical features of diabetic retinopathy are; 'microaneurysms, retinal haemorrhages, exudates, cotton wool spots, neovascularisation, fibrosis, pre-retinal and vitreous haemorrhages'. These features occur in various combinations in different patients and are used to classify the severity of the disease.

2.3.4.5 Neuropathy

Diabetic neuropathy (DN) is a common disorder and is defined as signs and symptoms of peripheral nerve dysfunction in a patient with diabetes mellitus (DM) in whom other causes of peripheral nerve dysfunction have been excluded.

Clinical classifications of diabetic neuropathies.are,

Symmetric

- Diabetic polyneuropathy
- Painful autonomic neuropathy
- Painful distal neuropathy with weight loss “diabetic cachexia”
- Insulin neuritis
- Polyneuropathy after ketoacidosis
- Polyneuropathy with glucose impairment
- Chronic inflammatory demyelinating polyneuropathy with diabetes mellitus

Asymmetric

- Radiculoplexoneuropathies-

  -Lumbosacral

  -Thoracic

  -Cervical
- Mononeuropathies
- Median neuropathy at wrist
- Ulnar neuropathy at the elbow
- Peroneal neuropathy at the fibular head
- Cranial neuropathy

Distal symmetrical polyneuropathy(DSPN)

DSPN is the commonest type of DN and probably accounts for 75% of DNs . Many physicians incorrectly presume that DSPN is synonymous with DN. It may be sensory or motor and may involve small or large fibres, or both. Sensory impairment occurs in glove and stocking distribution and motor signs are not prominent. Its is further classified into large fibre and small fibre neuropathy. Large fibre neuropathy is characterised by painless paresthesia with impairment of vibration, joint position, touch and pressure sensations, and loss of ankle reflex. In advanced stage, sensory ataxia may occur. Large fibre neuropathy results in slowing of nerve conduction,

impairment of quality of life, and activities of daily living. Small fibre neuropathy on the other hand is associated with pain, burning, and impairment of pain and temperature sensations, which are often associated with autonomic neuropathy.

Painfull Diabetic neuropathy

About 10% of diabetic patients experience persistent pain. Pain in DN can be spontaneous or stimulus induced, severe or intractable. DN pain is typically worse at night and can be described as burning, pins and needles, shooting, aching, jabbing, sharp, cramping, tingling, cold, or allodynia. Some patients develop predominantly small fibre neuropathy manifesting with pain and paresthesia early in the course of diabetes that may be associated with insulin therapy (insulin neuritis).

Diabetic autonomic neuropathy

Diabetic autonomic neuropathy affects various organs of the body resulting in cardiovascular, gastrointestinal, urinary, sweating, pupils, and metabolic disturbances. Because of diversity of symptoms, autonomic DN often goes unnoticed by both the patient and the physician. Autonomic nerve involvement can occur as early as one year after the diagnosis of DM. Diabetic autonomic neuropathy usually correlates with severity of somatic neuropathy.

Asymmetrical proximal diabetic neuropathy

It is also referred to as diabetic amyotrophy but should better be called as diabetic proximal neuropathy. The other examples of proximal DN include thoracic radiculopathy and proximal diffuse lower extremity weakness that should be grouped under a single term diabetic polyradiculopathy, as these are diverse manifestations of same phenomena; root or proximal nerve involvement. The weakness of pelvic femoral muscles occurs abruptly in a stepwise manner in the people above 50 years of age.

Most of these patients have NIDDM but it is unrelated to the severity or duration of diabetes. The patients complain of pain in low back, hip, anterior thigh, typically unilateral but may be bilateral. Within days or weeks, the weakness and wasting of thigh and leg muscles follows . Knee reflex is reduced or absent. Numbness or paresthesia is minor phenomena. Weight loss occurs in more than half the patients

Limb neuropathies

There are two major mechanisms of limb neuropathies in diabetics: nerve infarction and entrapment. Nerve infarctions are associated with abrupt onset pain followed by variable weakness and atrophy. As the primary pathology is axonal degeneration, the recovery is slow over a period of months. Median, ulnar, and peroneal nerves are most commonly affected.

Mononeuropathy

In diabetic patients, nerve entrapment is commoner than nerve infarction. The entrapment neuropathies have insidious onset, have characteristic electro diagnostic features such as conduction block or segmental nerve conduction slowing in the entrapped segment of the nerve. Carpal tunnel syndrome is three times more common in diabetic patients than the normal population. The other entrapment neuropathies in diabetic patients are ulnar, radial, lateral femoral cutaneous nerve of thigh, peroneal and medial and lateral planter nerves.

Cranial neuropathy

Cranial neuropathy in diabetic patients, most commonly involve the oculomotor nerve followed by trochlear and facial nerve in order of frequency. Third nerve palsy with pupillary sparing is the hallmark of diabetic oculomotor palsy and is attributed to nerve infarction. The pupillary fibres are peripherally located; therefore escape in diabetic oculomotor palsy.

Multiple neuropathies

Multiple neuropathies refer to the involvement of two or more nerves. As in mononeuropathy the onset is abrupt in one nerve and occurs earlier than the other nerves, which are involved sequentially or irregularly. Nerve infarctions occur because of occlusion of vasa nervosum and should be differentiated from systemic vasculitis.

Diagnosis of diabetic neuropaty

The American Academy of Neurology recommends that DN is diagnosed At least one of each of the five criteria is needed:

1. Symptoms

2. Signs

3. Electrodiagnostic tests

4.Quantitative sensory

5.Autonomic testing.

This may be necessary in research protocols. However. in clinical practice two of five criteria have been recommended.

Nerve conduction studies

Motor nerve conduction, F response, and sensory nerve conduction studies are important methods of documentation and follow up of nerve functions in DN. Motor nerve conduction studies are affected in a small subset of DN (large fibre neuropathies). Even in large diameter fibre neuropathy nerve conduction velocity (NCV) is insensitive for many pathological changes known to be associated with DN.The nerve conduction changes are non- specific and key to the diagnosis lies in excluding other causes or those superimposed on DN. Entrapment neuropathies are common in diabetic patients and result in unilateral NCV changes, especially across the entrapped segment of the nerve. The commonest abnormality in diabetes is reduction in the amplitude of motor or sensory action potentials because of axonopathy. Pronounced slowing of NCV suggests demyelinating neuropathy, which is rarely associated with diabetes; therefore pronounced slowing of NCV in a diabetic patients should prompt investigations for an alternative diagnosis. However, the likelyhood of CIDP occurring in diabetic patients is 11 times higher than the normal population. The NCV is gradually diminished in DN, with estimates of a loss of about 0.5 m/s/y.

Pathogenesis

The cause of DN though remains unknown but ischaemic and metabolic components are implicated. Hyperglycaemia induces rheological changes, which increases endothelial vascular resistance and reduces nerve blood flow. Hyperglycaemia also causes depletion of nerve myoinositol through a competitive uptake mechanism. Moreover, activation of polyol pathway in the nerve through enzyme aldose reductase leads to accumulation of sorbitol and fructose in the nerve

and induces non- enzymatic glycosylation of structural nerve proteins. Hyperglycaemia also induces oxidative stress. Activation of protein kinase C has been linked to vascular damage in DN. These changes result in abnormal neuronal, axonal, and Schwann cell metabolism, which result in impaired axonal transport. Direct measurement of glucose, sorbitol, and fructose in nerves of diabetic patients showed correlation with the severity of neuropathy.Endoneural hypoxia is produced by increased vascular resistance and reduced blood flow in the nerve. Hypoxia leads to further capillary damage, which in turn aggravates disturbance in axonal transport and reduced Na- K ATPase activity leading to axonal atrophy and impairment of nerve conduction.

2.3.5 Diabetic dyslipidemia

The defects in insulin action and hyperglycemia could lead to dyslipidemia in patients with diabetes. In the case of T2DM, the obesity/insulin resistant state that is at the basis of the development of this disease can in itself lead to lipid abnormalities independently of hyperglycemia. In poorly controlled T1DM hypertriglyceridemia and reduced HDL-commonly occurred.

2.3.6 Glycated Haemoglobin (HbA1C)

HbA1c was introduced into clinical use in the 1980s and subsequently has become a cornerstone of clinical practice. HbA1C reflects average plasma glucose over the previous eight to 12 weeks.It can be performed at any time of the day and does not require any special preparation such as fasting. These properties have made it the preferred test for assessing glycaemic control in people with diabetes. More recently, there has been substantial interest in using it as a diagnostic test for diabetes and as a screening test for persons at high risk of diabetes.The International Expert Committee recommended the use of HbA1C to diagnose diabetes mellitus with a threshold > 6.5%.However, the diagnosis should be confirmed by a repeat test unless symptoms of hyperglycaemia and blood glucose level of >11.1 mmol/l (>200mg/dl) are available.In addition, those with an HbA1C level between 6 and 6.5% have been identified as being at very high risk of developing diabetes, and the risk increases substantially as the values increase.

CHAPTER-3
MATERIALS AND METHODS

3.1 STUDY AREA AND SETTING

The study period is covered from June 2017 to July 2019 at the Govt. Siddha Medical College and Hospital, Palayamkottai- 627 002, Tirunelveli, Tamil Nadu. All procedures were carriedout before getting the permission from Instutional Ethical Commitee.

3.2 STUDY DESIGN

The study design is a prospective open labelled non randomized Phase-II clinical trial of 40 type-II diabetic study. The included selection were newly diagnosed and already diagnosed as type-II diabetic patients with or without taking treatment. A written informed consent form was recruited in the study. The purpose of the study was explained to the patients before administration of trial drug. The patients information, life style, anthropometric measurements and Siddha parameters were recorded to starting the treatment.

The total number 40 patients, equally in both sex and age between 40 to 70 were taken for this study. The selected patients were treated the trial drug at end of the study period (90 days).

3.3 SELECTION OF PATIENTS

After inclusion of cases is screening before starting the treatment. The criteria for selection of patients is already diagnosed in type 2 Diabetes (fasting, post prandial,Random blood sugar, high range of HbA1C).

Detailed personal history, family history, occupation, habits, clinical symptoms, medical history, and the duration of illness were recorded in all patients (Proforma annexed).

3.3.1 Inclusion Criteria

1. Age between 40 and 70 years
2. Type-II diabetes mellitus

   If yes in any of three, FBS - > 126 mg/dl or PPBS - > 200mg/dl or HbA1C > 6.5 and<10 (ADA 2017).
3. Total cholesterol - >199 mg/dl or LDL - >159 mg/ dl or Triglycerides - >150 mg/dl or HDL level - < 60 mg /dl
4. Willing to give blood sample for the Lab investigations
5. Diabetic Neuropathy Pain Score greater than 4/10

3.3.2 Exclusion Criteria

1. Age below 40 and above 70
2. If yes in any one of three

   FBS - < 125 mg/dl or PPBS - <199mg/dl or HbA1C < 6.5
3. High lipid profile, dyslipidaemia has values of, Total cholesterol <199 mg/dl or LDL<159 mg/ dl or Triglycerides <150 mg/dl
4. Type-I diabetes mellitus
5. Secondary hypertension
6. Pregnant woman
7. Lactating mother
8. Chronic kidney disease / Renal failure
9. Corticosteroid therapy
10. Chronic active viral hepatitis/cirrhosis/ascites
11. Endocranial disorder
12. Femoral/Sciatic Nerve dysfunction
13. Demyelinating polyneuropathy
14. Mechanical and Traumatic nerve injury/Compressive mylo radiculopathy

3.3.3 Diagnosis

The Siddha diagnostic procedure were included this study, which are,

- Poriyal Arithal
- Pulanal Arithal
- Vinathal
- Mukkutra nilaigal

- Envagai thervugal
- Nilam
- Kaalam & Udal kattugal

3.3.4 Investigations

Blood:

- TC, DC, ESR, Hb%
- Blood Sugar (Fasting and Post Prandial), Serum Urea, Serum Creatinine
- HbA1C
- Fasting lipid profile

Urine:

- Albumin, Sugar, Deposits

The Biochemical Analysis were carried out before and after administration of trail drug.The blood sugar and other lab investication have monitored once in twenty days.

3.4 TREATMENT

3.4.1 Preparation of Trial Medicine (Annexure-I)

The herbal preparation of kadukkai chooranam have been selected from the Classical Siddha literature. ***Reference: Gunapadam Mooligai Muthal Pagam Page No.201***

Kadukkai, Vallaraiyin Thanimanbu Pg.No. 9

3.4.2Collection and authentication of TrialMedicine (Annexure-II)

The *Terminalia chebulla* fully matured fruits was collected from the Nagar kovil Siddha medical shop, Tamilnadu. The fruits will be identified and authenticated by the Medicinal Botanist and Gunapadam experts at Government Siddha College and Hospital, Palayamkottai - 627002..

3.4.3 Preclinical Analysis of Trial Medicine

All the preclinical studies of the study drug which havebeen included in Bio chemical and pharmacological studies and cross checked before starting the treatment.The Biochemical analysis were done in Dept. of Biochemistry, GSMCH, Palyamkottai.

The pharmacological activities- Anti- hyperglycaemic, Anti-Hyperlipedemic, Acute and sub-acute toxicity,Analgesic and Anti microbial activities were carried out in this study. studies were done in K.M. College of Pharmacy, Madurai -625107.

3.4.4 Ethical Review

The study was conducted in accordance with the ethical principles that are consistent with Good Clinical Practice guidelines and obtained prior approvals before start of the trial from the Institutional Ethics Committee of GSMCH, Palayamkottai (**GSMC-IV-IEC/2017/Br.-I/08/29.05.2017**) and Institutional animal ethical committee (IEAC) of K.M. College of Pharmacy, Madurai (**TNMGRMU/KMCP/IEAC/19/2018**). The trial was applied and approved by the Clinical Trial Registry of India.

3.4.5 Study Enrolment

Participants were informed in Tamil language, regarding the trial, the expected benefits and their right to opt-out of trial at any time without pre judice. Informed written consent was obtained from each participant, prior to his/her inclusion into the trial.

Before commencing the trial all the subjects were advised diet based on their body mass index, but no recommendations on diet were given during trial period. All subjects of age range between 40-70 years with Fasting Plasma Glucose (FPG) >126mg/dl and two-hour Postprandial Plasma Glucose (PPPG) between >200mg/dl and with lipid profile Total cholesterol->199 mg/dl, LDL->159 mg/ dl, Triglycerides ->150 mg/dl and HDL level - < 60 mg /dlwere included in the study.

The subjects with history of serious adverse effects or hypersensitivity reactions to the medication such as rashes, diarrhoea, vomiting etc., and history of treatment with other anti-hyperglycaemic drugs, active liver disease or hepatic dysfunctions, higher serum creatinine (> 2.5 mg/dl) and serious or unstable medical or psychological condition are excluded from the study.

During the visit, body weight, blood pressure, cardiovascular,neurological and respiratory system were clinically recorded . During the treatment any adrverse reaction or side effects of patients, immediately to inform patient and pharmacovigilance committee. At the end of the study period, all the patients were instructed to follow diet control, regular exercise, yoga, meditation and to monitor

their blood sugar levels, HbA1C, and lipid levels periodically. They were also advised to pursue the further treatment in the PG, Pothu Maruthuvam OP for the follow up study.

3.4.6 Statistical Analysis

All data were analysed using the SPSS 20.0 (IBM). Data were expressed as means and standard deviation. The significance of the difference between the means of the baseline and the final examinations was tested using the paired "t" test. A probability value of <0.05 was considered to be statistically significant.

. CHAPTER IV

RESULT AND OBSERVATION

4.1 PRE CLINICAL STUDY

4.1.1 ANTI HYPERGLYCEMIC:

Table no: 4.1.2a is showed, the levels of initial and final blood glucose, and change in body weight, in normal rat, and treatment control animals in each group. The mean body weight of diabetic rats (G2) was significantly decreased as compared to normal control rats. The body weight of diabetic control rats treated with siddha formulation KADUKKAI CHOORANAMat a dose of 100 and 200mg/kg was increased the body weight non-significantly as compared to normal control animals.

Table No: 4.1.2.a - Effect of siddha **formulation *KADUKKAI CHOORANAM* on initial and final body weight and blood glucose in normal and treated animals**

GROUP	Body weight (g)		Blood glucose (mg / 100ml) Initial	Blood glucose (mg / 100ml) Final
	Initial	Final		
G1	230 ± 7.15	241± 7.35	89.55 ± 3.37	91.78 ± 3.85
G2	231 ± 6.68	176 ± 4.50**(a)	89.78 ± 3.45	219.48 ± 6.96**(a)
G3	237 ± 7.30	240 ± 7.30	89.70 ± 4.20	125.38 ± 4.35**(b)
G4	231± 7.32	240 ± 7.38	83.77± 3.65	141.43± 5.32**(b)
G5	231 ± 7.35	244 ± 7.45	96.42 ± 3.78	131.49 ± 4.45**(b)

As per table 4.1.2;b the blood sugar level was showed after useing the test control dose 100, 200 mg of KC administrated in Wister albino Rats. The control group was compaired in standard (Group III) and treatment control (Group IV &V). The above table shows 100 and 200 mg of KC was compaired group II &III.

The blood sugar and lipid was rdecreased. The conclusion of the above results refined significantly decreased blood sugar level. Standarded and control result showed, initial blood glucose level was 89.70 ± 4.20 and final glucose level was 125.38 ± 4.35**(b) ; treatment control (& IV &V) showed initial blood glucose level 96.42 ± 3.78 final glucose level 131.49 ± 4.45**(b).

Table no: 4.1.2.b; Effect of siddha formulation *KADUKKAI CHOORANAM* on plasma insulin, Hemoglobin & Glycosylated hemoglobin in normal and treated animals.

GROUPS	Haemoglobin (gm/100ml)	Glycosylated haemoglobin HbA_1 (%)	Plasma Insulin (µU/ml)
G1	12.85 ± 1.65	0.45 ±0.06	38.57 ± 2.84
G2	6.27 ± 0.79**(a)	0.99 ±0.18**(a)	13.85 ± 1.85**(a)
G3	14.2 ± 1.46**(b)	0.43 ±0.08**(b)	29.48 ± 2.48**(b)
G4	12.75 ± 0.87**(b)	0.49 ±0.16**(b)	26.76 ±2.45**(b)
G5	11.98 ± 1.22**(b)	0.45±0.06**(b)	28.95± 2.76**(b)

- Values are expressed as mean ± SEM.
- Values were compared by using analysis of variance (ANOVA) followed by Newman-Keul's multiple range tests.
- ** (a) Values are significantly different from normal control G1 at P<0.001.

** (b) Values are significantly different from Diabetic control G2 at P<0.

As per table 4.1.2;b the HBA1-C was showed standard and 100, 200 mg of KC administrated in Wister albino Rats. The control group was compaired in standard and treatment control (Group IV &V). The above table was showed 100 and 200 mg of KC, it was reduced the HBA1-C level in standard control (gRoup III). The conclusion of the above results revealed that, the significantly reduced HBA1-C. The standard control results showed haemoglobin level 14.2 ± 1.46**(b) , HbA1-C level 0.43 ±0.08**(b) ; plasma insulin level - 29.48 ± 2.48**(b), was decreased in compared in treatment and control groups (Haemoglobin - 11.98 ± 1.22**(b); HbA1-C - 0.45±0.06**(b); plasma insulin level - 28.95± 2.76**(b)) .

4.1.2 ANTI HYPERLIPIDEMIC ACTIVITY

Table 4.1.2;a: Effect on siddha formulation *KADUKKAI CHOORANAM* in Lipid Profile

GROUPS	Total cholesterol (mg/dl)	Triglycerides (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/d)	AI	LDL/ HDL
Normal Control	49.70 ± 1.70	57.10 ± 0.90	26.45 ± 1.18	14.30 ± 0.76	30.86 ± 1.05	0.87 ± 0.50	0.54 ±
Cholesterol Control	115.40 ± 1.56**(a)	161.50 ± 1.68**(a)	11.90 ± 0.65**(a)	30.96 ± 1.32**(a)	13.05 ± 0.70**(a)	8.51 ± 1.33**(a)	2.60 **(a)
Standard Control	72.60 ± 1.42**(b)	82.10 ± 1.80**(b)	21.5 ± 0.40**(b)	20.05 ± 0.76**(b)	24.85 ± 0.76**(b)	2.34 ± 2.33**(b)	0.92 **(b)
Treatment control (100mg)	92.86 ± 1.24**(b)	114.40 ± 1.96**(b)	17.0 ± 0.50**(b)	25.25 ± 0.60**(b)	17.84 ± 0.42**(b)	4.39 ± 1.48**(b)	1.46**(b)
Treatment control (200mg)	83.85 ± 0.94**(b)	96.5 ± 1.10**(b)	20.30 ± 1.28**(b)	22.12 ± 0.72**(b)	21.25 ± 0.52**(b)	3.13 ± 0.24**(b)	1.08 **(b)

RESULTS

The reduction of plasma total cholesterol was associated with a decrease in its LDL fraction which is a major, potentially modifiable risk factor of cardio vascular disease and the target of drug. Many suggest that the cholesterol lowering activity of this product appears to be due to the enhancement of LDL – C catabolism through hepatic receptors (C.Vijaya et al.2009). In addition siddha formulation *KADUKKAI CHOORANAM*showed protective action which is reported to have a preventive function against atherogenesis since an independent inverse relationship between blood HDL – C levels and cardio vascular risk incidence is reported.(T.Chidambaram et al.2007) .The mechanism of reduced hyperlipidemia is the enhancement of Lecithin Cholesterol Acyl Transferase (LCAT) and inhibition of Hepatic Triglyceride Lipase (HTL) on HDL which may lead to a rapid catabolism of blood lipids through entero hepatic tissues. (A.E. Ahire et al. 2005),(L.Anila et al.2002). It reported that triglycerides (M.A.Austin et al.1984) layed a key role in the regulation of lipoprotein interaction to maintain normal lipid metabolism. Indeed, the elevated plasma TG levels were associated with an increased .incidence of coronary artery disease . More

over these higher plasma TG levels have been attributed mainly to increase population of small, dense LDL deposits which are very atherogenic and enhanced cholesteryl ester mass transfer from apolipoprotein containing lipoproteins (VLDL , LDL and TG) has also been proposed to be major determined of cholesteryl esterification, its transfer and HDL remodeling in human plasma. Administration of siddha formulation *KADUKKAI CHOORANAM*(KC) is provides a beneficial action on rat lipid metabolism with regard to the reduction of AI. Infarct, the AI was decreased in all treated groups. Similar results were reported by others when studying the hypolipidemic effects of natural products. This ameliorative action was due to the plasma lipid lowering activity of different constituents of the formulation.

As for table 4.1.1;a the lipid profile was showed stamdard and 100, 200 mg of KC administrated in Wister albino Rats. The cholesterol control was compaired in standard and treatment control. The above table shows 100 and 200 mg of KC, it was compaired to reduced the lipid level in standard control. The conclusion of the above results refined significantly reduced lipid profile.

Standard control showed : Total cholesterol - **72.60± 1.42**$^{**(b)}$; Tri glycerides - **82.10 ± 1.80**$^{**(b)}$; HDL - **21.5 ± 0.40**$^{**(b)}$; LDL - **20.05 ± 0.76**$^{**(b)}$; VLDL - **24.85 ± 0.76**$^{**(b)}$; VI -**2.34 ± 2.33**$^{**(b)}$; LDL/HDl -**0.92** $^{**(b)}$, and Treatment control showed ; Total cholesterol - **83.85 ± 0.94**$^{**(b)}$; Tri glycerides - **96.5 ± 1.10**$^{**(b)}$; HDL - **20.30 ± 1.28**$^{**(b))}$; LDL - **22.12 ± 0.72**$^{**(b)}$; VLDL - **21.25 ± 0.52**$^{**(b)}$; VI -**3.13 ± 0.24**$^{**(b)}$; LDL/HDl - **1.08** $^{**(b)}$. The Values are found\ by using ONE WAY ANOVA followed by Newman Keul's multiple range tests.** (a) values were significantly different from normal control at P< 0.01.** (b) Values were significantly different from hyperlidemic control at P< 0.01. In Table no 4.1.1;a. the end of result found, after the treatment of Kadukkai choornam, TC, Triglycerides & LDL was decreased in treatment control (Group IV,V), it was compared Group I,II and III standard control .

4.1.3 PHYTOCHEMICAL STUDY:

The *KADUKKAI CHOORANAM*was subjected to qualitative chemical investigation details of the various tests performed for the presence of phyto constituents is shown in Table 4. 1.3.a. The structural characterization of the herbal drugs is need of the hour and the objective is to establish the elemental and structural characteristics of *KADUKKAI CHOORANAM*. Although, many research works has been done the herbal drugs.So, performed standardization, structural standardization, Physio and phytochemical parameters is very nesceessiate to stantardization of *KADUKKAI CHOORANAM*.

Table 4.1.3;a: Phyto chemical tests for *KADUKKAI CHOORANAM*

TEST	**INFERENCE**	
Mayer's test Dragendroff's test Hager's test	**Alkaloids**	Present Present Present
Molisch test Legal's test Borntrager's test for anthraquinones	**Carbohydrates and Glycosides**	Present Present Present
Liebermann-Burchard test Salkowski test	**Phytosterols**	Present Present
Shinoda test Magnesium turnings and hydrochloric acid Fluorescence test	**Flavanoids**	Present Present Present
Ferric chloride test Potassium dichromate test Lead acetate test	**TANNINS**	Present Present Present
Millon's test Biuret test	**PROTEINS**	Present Present

Ninhydrin test		Present
Spot test Saponification test	**Fixed oils and fats**	Abscent Abscent
Phloroglucinol test	**Lignin**	Present
Frothing test	**Saponins**	Abscent

Above the 4.1.3.a results are, the presence of phytochemical Alkaloids, Carbohydrates and Glycosides, Phytosterols. Flavanoids, Tannins, Proteins and Fixed oils and Fats, Lignin, Saponins are absence from this study.

4.1.4 ANALGESIC ACTIVITY

Acetic acid-induced writhing response

The different doses of siddha formulation KADUKKAI CHOORANAMhad significant analgesic effects in animals models.The results of 100 and 200 mg/Kg of kadukkai choornam waa significantly reduced,it was compared the modern medicine Diclofenac sodium.(Table 4.1.4;a).

Table No;4.1.4;a- Effects of siddha formulation *KADUKKAI CHOORANAM* on acetic acid–induced writhing response (N=6 in each group).

Groups	Treatment	(number of writhing movements) (Mean ± S.E)	Percentage %
Group I	Distilled water	38.5±2.8	-
Group II	Diclofenac sodium 10mg/kg	6.5±0.6	83.11%**
Group III	100mg/kg KADUKKAI CHOORANAM	12.0±1.3	68.83%**
Group IV	200mg/kg KADUKKAI CHOORANAM	11.8±1.0	69.35%**

- Values are expressed as mean ± SEM.

  * (b) Values are significantly different from Toxic control G2 at P<0.01.

As for table 4.1.4;a The above table showed, 100 and 200 mg of KC, it was reduced the Pain level compared in standard groups. The Number of writhing movements 6.5±0.6) and treatment groups writhing movements was 11.8±1.0HbA1-C - 0.45±0.06**(b);

Discussion

The analgesic activity was assessed by writhing test which has been reported to be useful for investigation of peripheral antinociceptive activity and performed as a chemical pain model (IV, V). The siddha formulation *KADUKKAI CHOORANAM* demonstrated a dose-dependent, significant antinociceptive activity in animal models of pain. Acetic acid believed to increase the PGE2 and PGF2α in peritoneal fluid (6). The analgesic activity shown in models of pain is indicative that siddha formulation KADUKKAI CHOORANAMmight possess centrally and peripherally mediated antinociceptive properties.

Chemical components of siddha formulation KADUKKAI CHOORANAMsuch as flavonoids, saponins or phenolic compounds may be responsible for the antinociceptive activities of this formulation. Since the findings of this study revealed a significant analgesic effect of the siddha formulation KADUKKAI CHOORANAM, it can be concluded that terpenoids and specially saponins of siddha formulation KADUKKAI CHOORANAMmay be responsible for the observed analgesic effect which should be proved by further investigations.

4.1.5 TOXICITY STUDY

4.1.5.1 Acute Toxicity study for *Kadukkai Chooranam*:

In the acute toxicity study was carried out the female and male and albino mice. All the study animals are feded by oral dose 50, 100,200 and 400 mg of *KADUKKAI CHOORANAM* for 60 days. At the end of the study, the animals were sacrificed and assessed by the effect of KADUKKAI CHOORANAM. The body weight and relative organs, hematological, biochemical and histopathological parameters are noted. The Acute toxicity and mortality ratio was given in Table 4.1.5a.

Table No: 4.1.5;a;- Sign of Acute toxicity and mortality ratio :

	Dose ($mg.kg^{-1}$)	Sign of Toxicity ($ST.NB^{-1}$)	Mortality ($D.S^{-1}$)
Group I	0	0/3	0/3
Group II	300	0/3	0/3
Group III	2000	0/3	0/3

ST- sign of toxicity; NB- normal behavior; D- died; S- survive. Values are expressed as number of animals (n=3).

There was no mortality or morbidity were observed in three group of animals, during 14-days period in single daily dose administered 50, 300, 2000 mg/kg/bw (Table-4.1.5.a). The animals did not shows no changes in the general physical appearance during the observation period. Morphological characteristics such as skin, eyes and nose were appeared in normal and no tremors, convulsion, salivation, diarrhea, lethargy or unusual behaviors were observed. Gait and posture, reactivity to handling or sensory stimuli, grip strength was also normal in state.

4.1.5.2; SUB ACUTE TOXICITY STUDY

The effect of KC was observed, the effect on the body weight changes, significant increase ($p<0.05$) in body weight in all the treated animals were observed. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05(Table 4.1.5. b).

Table4.1.5.b The Effect on Sub acute study in body weight

Treatment	**Day 1**	**Day 07**	**Day 14**	**Day 28**
Control	185.15±6.8	185.45 ±6.20	194.15 ±6.35	194.7±6.58
KC **50 $mg.kg^{-1}$**	192.30 ±6.4	191.30 ±6.30	196.25 ±6.70	196.30±6.72*
KC 100 $mg.kg^{-1}$	184.35 ±5.7	187.30 ±6.40	194.55 ±7.10	195.36±6.30*
KC 200 $mg.kg^{-1}$	193.30 ±7.2	193.15±6.50	193.90 ±7.20**	204.45±7.26**
KC 400 $mg.kg^{-1}$	185.65 ±6.05	190.15 ±5.60	193.60 ±6.35**	205.66±7.38**

The Sub acute Effect of KADUKKAI CHOORANAM on internal organs in rats

The effects of KC on kidney, heart, liver and brain of the rats were recorded. From the significant ($p<0.01$) changes in the weights of various organs of the animals occurred with higher doses of the extract (400 $mg.kg^{-1}$ bwt), but macroscopic examinations visualized no changes in color of the organs of the treated animals compared with the control group. The statistical analysis was carried out using one way ANOVA method, where **P<0.01.The results are expressed in Table.4.1.5.c.

Table No.4.1.5.c. Changes in internal organs

Treatment	Heart (gms)	Kidney (gms)	Liver (gms)	Brain (gms)
Control	0.34 ± 0.05	0.64± 0.03	3.30± 0.05	0.65± 0.05
KC 50 $mg.kg^{-1}$	0.35± 0.02	0.80± 0.03	3.42± 0.03	0.68± 0.3
KC 100 $mg.kg^{-1}$	0.36± 0.06	0.78± 0.04	3.34±0.02	0.66± 0.2
KC 200 $mg.kg^{-1}$	0.35± 0.04	0.73± 0.02	3.32± 0.02	0.73± 0.05
KC 400 $mg.kg^{-1}$	0.34± 0.03	0.74± 0.03	3.35± 0.03	0.75± 0.05

Sub acute effect of *KADUKKAI CHOORANAM* on lipid profile in rats

Table.4.1.5.dThe effect of KC on biochemical parameters .

Treatment	**Glucose ($mg.dl^{-1}$)**	**Cholesterol ($mg.dl^{-1}$)**	**Triglyceride ($mg.dl^{-1}$)**	**HDL ($mg.dl^{-1}$)**	**LDL ($mg.dl^{-1}$)**
Control	92.65± 0.62	37.62± 0.56	26.25± 0.45	133.25± 0.55	82.15±1.72
KC 50 $mg.kg^{-1}$	90.50± 0.56	23.85± 0.25*	11.22± 0.23*	173.28±0.65*	69.59±1.28
KC 100 $mg.kg^{-1}$	87.45± 0.47	24.74± 0.26*	13.42± 0.28*	163.18±0.78*	67.84±1.10
KC 200 $mg.kg^{-1}$	88.25±0.55**	31.18± 0.30	15.84± 0.38*	182.30±0.84*	46.60±1.30
KC 400 $mg.kg^{-1}$	84.25±0.45**	30.78± 0.28	17.28± 0.34*	180.2± 0.85*	44.50±0.84

The effect of **KC** on various biochemical parameters of the experimental animal 'rats' were tested. From the study it was evident that, there was significant decrease ($p<0.05$) in the plasma glucose level in treated rats especially at higher dose (400 $mg.kg^{-1}$) compared with control rats. The control rats were administered only with 5 ml of normal saline. Significant decrease ($p<0.05$) in the plasma total cholesterol (TC), triglyceride (TG) and LDL-cholesterol levels were observed. But a significant increase ($p<0.05$) in HDL-cholesterol levels were observed in all the treated animals compared with the control studies, AST, ALT and ALP levels were also normal in the KC used animals. From the results of biochemical study there was no evidence of Sub acute toxicity associated with the administration of higher concentration of KC.

Changes in hepatic enzymes

Table 4.1.5e .The AST, ALT, ALP, TP and Albumin values was compared in Group I and other groups II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where determined **P<0.01 *P<0.05.

Table.4.1,5.e; The results and effects of KC on biochemical parameters such as AST, ALT, ALP, TP and Albumin in rats are,

Treatment	AST ($IU.l^{-1}$)	ALT ($IU.l^{-1}$)	ALP ($IU.l^{-1}$)	TP ($g.l^{-1}$)	ALBUMIN ($g.l^{-1}$)
Control	326.5±12.40	69.5± 3.18	251.58± 8.80	67.85± 3.32	37.15±2.35
KC 50 $mg.kg^{-1}$	315.0±9.50**	67.5± 2.20**	264.10± 2.75**	68.30± 2.32	34.30±2.65
KC 100 $mg.kg^{-1}$	316.3±7.20**	65.1± 3.15**	258.18± 6.70**	78.15± 2.82	36.30±3.05
KC 200 $mg.kg^{-1}$	311.4±7.95	60.4± 2.90	263.00± 5.20	67.25± 3.32	38.20±2.75
KC 400 $mg.kg^{-1}$	321.2± 8.20	62.3± 3.52	267.40± 4.40	72.05± 2.58	37.48±2.70

Effect of KADUKKAI CHOORANAM on Hematological parameters in rats;

The effects of **KC** were observed for its effect on hematological parameters in experimental rat. Final study, a significant increased ($p<0.01$) in the haemoglobin,RBC values are increased after treated groups.There was no significant

changes in the calcium level in all the treated animals compared to the control. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV and V. The statistical analysis was carried out using one way ANOVA method, where *P<0.05 (Table.4.1.5.f).

Table.4.1.5.f The effect of KADUKKAI CHOORANAM on hematological parameters

Treatment	Haemoglobin ($g.dl^{-1}$)	RBC (10^6 /mm^3)	WBC (10^6 /mm^3)	Calcium ($mg.dl^{-1}$)
Control	13.3± 0.25	9.10± 0.02	11.4± 0.05	9.40 ±0.02
KC 50 $mg.kg^{-1}$	14.5± 0.26*	9.45± 0.04*	9.5± 0.01*	9.16 ±0.02
KC 100 $mg.kg^{-1}$	14.3± 0.15*	9.50± 0.02*	8.3± 0.32*	9.22 ±0.20
KC 200 $mg.kg^{-1}$	12.7± 0.20*	8.28± 0.12*	11.4± 0.03*	9.56 ±0.13
KC 400 $mg.kg^{-1}$	13.5± 0.35*	8.46± 0.45*	10.5± 0.13*	9.70 ±0.02

DISCUSSION

The acute toxicity study of KADUKKAI CHOORANAM(KC)was carried out as per OECD-423 guidelines, no mortality was observed in control group as well as treated groups animals,maximum dose of 2000 $mg.kg^{-1}$ was not produced toxicity. Hence, $1/10^{th}$ of 2000 $mgkg^{-1}$ i.e. 200 $mg.kg^{-1}$ of dose was selected as a minimum dose for acute toxicity study. The results of sub acute toxicity study showed that treated animals were normal in growth pattern and body weight compared with control rats treated with normal saline. So, the changes in body weight can be used as an indicator of adverse effects of drugs and chemicals. Serum cholesterol and proteins mainly regulated via synthesis in the liver and increase or decrease in serum concentrations of constituents suggest liver toxicity. All test animals were subjected to gross necropsy .There was a slight decrease in plasma glucose level, when higher doses of **KC** (400 $mg.kg^{-1}$) were administered in the treated rats..The significant increase in the levels of hemoglobin (Hb) was found in treatment with KADUKKAI CHOORANAM(KC) with a higher dose of $200mgkg^{-1}$. The possible reason could be that one of the constituents KC may increase absorption of iron.

4.1.6: FTIR and EDAX ANALYSIS

4.1.6.1; Scanning electron microscopic study (SEM)

The Scanning electron microscopy is a complementary technique and shows the nature of kadukkaichooranam and its particle size(Bruneton,J., 1995). Pharmacognosy, Phytochemistry, Medicinal Plants)[7].Sample for SEM analysis were mounted on the specimen using carbon adhesive sheet. Small sample were mounted with 1sq. cm glass slide And kept in carbon adhesive sheet. Samples were coated with gold to a thickness of 100 AO using Hitachi vacuum evaporator. Coated sample were analysed in a Hitachi Scanning electron Microscope 3000 H model.

Elemental analysis by EDAX

EDAX was used for multiple sampling in various parts of the plant and can also provide information from an area of fewer manometers. This is very useful to characterize the crystals and other inclusions like trace elements present in the given sample KADUKKAI CHOORANAM(World Health Organization, Geneva, 2007).

4.1.6.2 Fourier Transform – Infra Red Spectroscopy Study (FTIR)

The FT-IR Spectrometer was carried out in KBr Pellet methods (Sathyanarayana B,2011) About 1/8th of the solid powder of Kadukkaichooranam was taken on a microspatula and about 0.25-0.50 teaspoons of KBr was added and thoroughly ground in an agate mortar with the pestle until Kadukkaichooranam became very fine. It was placed in a pellet die. The sample was pressed at 5000-10,000 psi and the sample was removed carefully from the die and placed in the FTIR sample holder (Chattopadhyay, R.R. and S.K. Bhattacharyya, 2007) . The computer was turned on and the software was launched and certain fine details of the working method were done. The sample was placed on Zn,Se crystal with a spatula until the pressure marker showed. The values are recorded.

Results and Discussion

The results of Scanning electron microscope in two different view and EDAX Trace elements profile & FT-IR data has compiled as follows,

Scanning Electron Microscope Analysis

The SEM picture (Fig. **4.1.6.a**) is under 1.00 KX resolution and the examining area of 800x800µm2 surface were taken for the samples (SarafASamant A 2013) . The surface of the sample is marked with cluster arrangement and in agglomerates form (Bruneton, J., 1995). The above SEM images showed average Particle Size ranges from 101 µm to 1115µm. In 100mu view, the surface of the sample grains is uniformly arranged in agglomerates. Particle Size ranges from 107 µm to 1244µm (Fig;**4.1.6.b**) and overall particles shape and angles are representd in Ferrets graph.

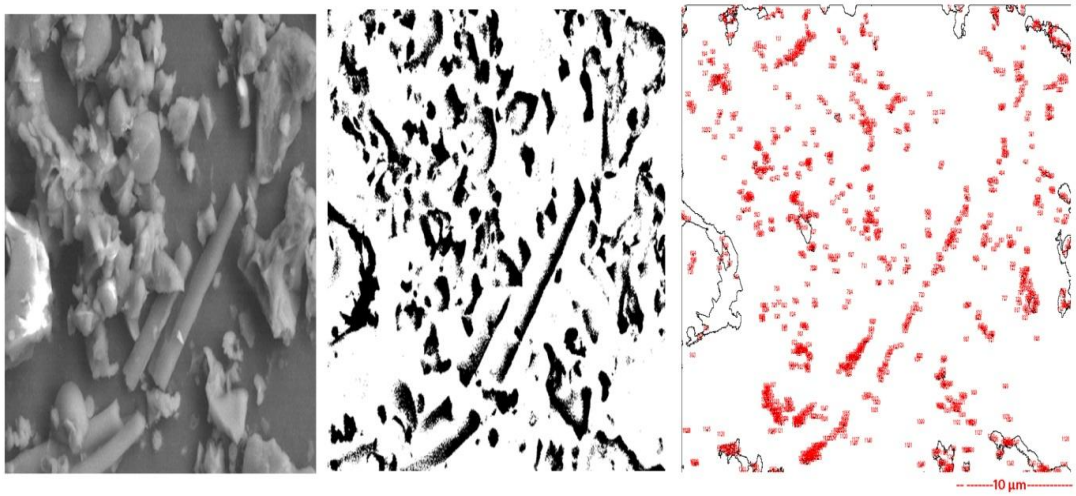


Figure 4.1.6.a. SEM image of Kadukkaisuranam of 10 µm view

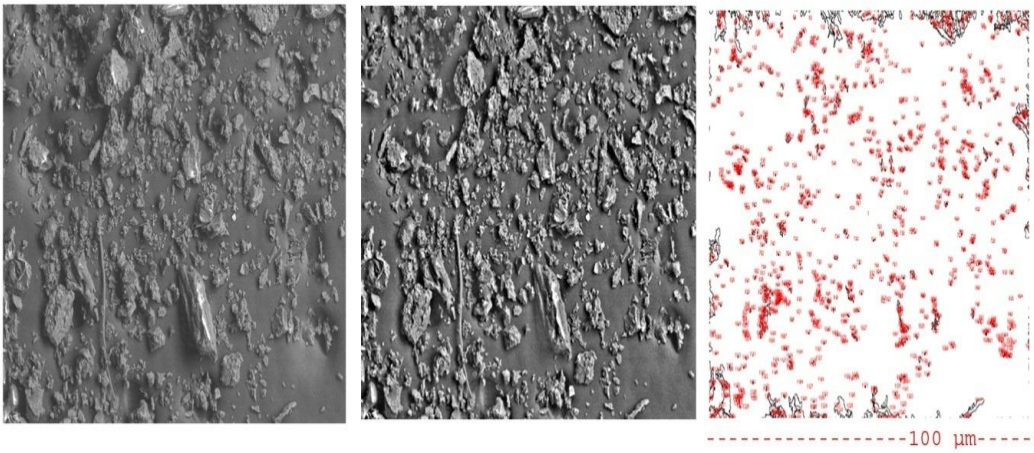


Figure.4.1.6.b; SEM image of Kadukkaichooranam of 100 µm view

Elemental Quantification of Kandukkaichooranam by EDAX

The elemental quantification of Kaddukaichooranam was done by EDAX method (Kesarla Mohan kumar et al,).The overall trace elements like Calcium, potassium has almost 4.3%, 2.5% of Oxygen and Potassium, 2.5% Magnesium & Hydrogen as the major compounds were found the results. **Figure 4.1.6.c. Identification of trance elements through EDAX**

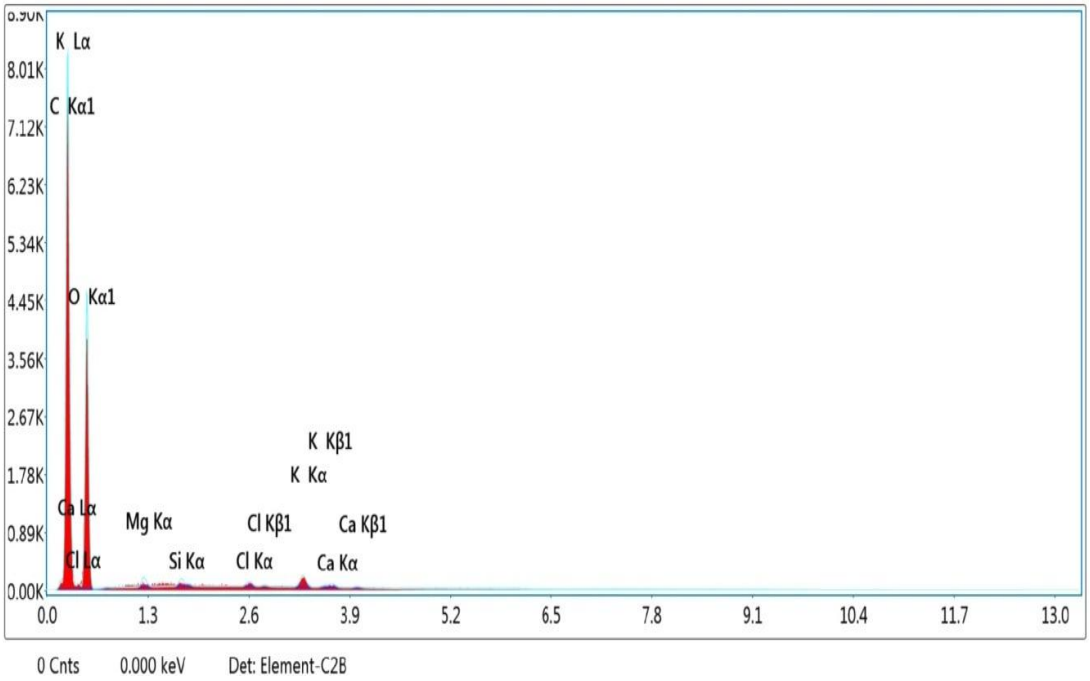


Figure.4.1.6.c Graaphical representation of EDAX profile of KADUKKAI CHOORANAM(in percentage)

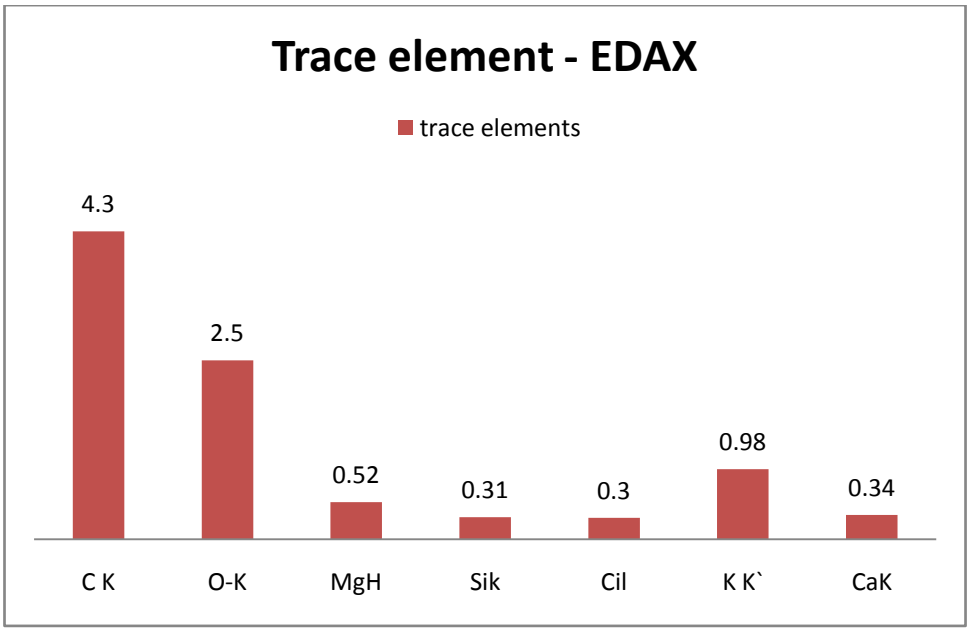


Figure ;4.1.6.d; FTIR Spectra of KadukkaiChooranam

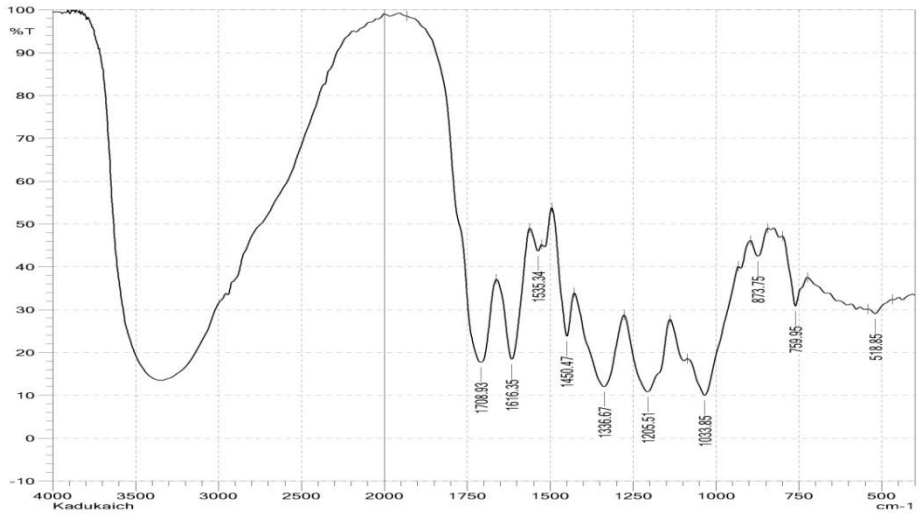


Table .4.1.6.A. FTIR observed Peak value of Kadukkaichooranam

Wave number (cm-1)	**Vibration modes of sample in IR region**	**Intensity of the bond**	**Functional groups**
1708.93	C=O Stretching	strong	conjugated aldehyde
1616.35	C=C stretching	Strong	α,β-unsaturated ketone
1535.34	N-O stretching	Strong	nitro compound
1450.47	C-H bending	Medium	Alkane
1336.67	S=O stretching	Strong	Sulphonamide
1205.51	C-O stretching	strong	alkyl aryl ether
1033.85	S=O stretching	Strong	Sulfoxide
873.75	C-Cl stretching	Strong	Halogen compounds
759.95	C=C bending	Strong	Alkene
518.85	C-Br bending	week	Alkyl

In FT-IR Spectra analysis, the specified drug Kadukkaichooranam has exhibits the FTIR Spectra values and peak value at 1708.93 has C=O Stretching , 1616.35 has C=C stretching, 1535.34 has N-O stretching, 1450.47 has C-H bending, 1336.67, 1205.51 has C-H bending, 1033.85 has S=O stretching, 873.75 has C-Cl stretching, 759.95 has C=C bending, 518.85 has C-Br bending.So,KC is contains conjugated aldehyde, α,β-unsaturated ketone, nitro compound, alkane, sulphonamide, alkyl aryl ether, sulfoxide, Halogen compounds, Alkene and Alkyl compounds were observed respectively(Ahn, M.J., C.Y. Kim, J.S. Lee, T.G. Kim and S.H. Kim et al., 2002).

4.2 .IN CLINICAL STUDY:

The results were observed with respect to the following criteria by clinical study on 20 out patients and 20 In patients of both sexes. The criteria were.

1. Distribution of Gender
2. Distribution of Age
3. Distribution of Educational
4. Distribution of Occupation
5. Distribution of Religion
6. Distribution of Marital Status
7. Distribution of Clinical Manifestation
8. Distribution of Duration of Illness
9. Distribution of Family History
10. Distribution of History of Previous Treatment of Madhumegam
11. Distribution of Personal History
12. Distribution of Socio-Economical Status
13. Distribution of Other System Involvement
14. Distribution of Body Mass Index
15. Distribution of Constitution of Body
16. Distribution of Gunam
17. Distribution of Kaalam
18. Distribution of Paruva Kaalam
19. Distribution of Thinai
20. Distribution of Mukkutram
    a). Derangement of Vatham
    b). Derangement of Pitham
    c). Derangement of Kapham
21. Distribution of Involvement of Udal Thathukkal
22. Distribution of Kanmenthiriyam
23. Distribution of Imporigal (Gnanendrium)
24. Distribution of Kosam
25. Distribution of Conditions of Envagai Thervugal
26. Distribution of Neer Kuri

27. Distribution of Nei Kuri
28. Distribution of Laboratory Analysis

a). HbA1C

b). Blood Glucose

c). Lipid Profile

29. Changes in diagnostic findings before and after treatment

a) Changes in Nerve conduction study and Neuropathic pain score
b) Changes in HbA1C, Blood Sugar and Lipid Levels before and after treatment

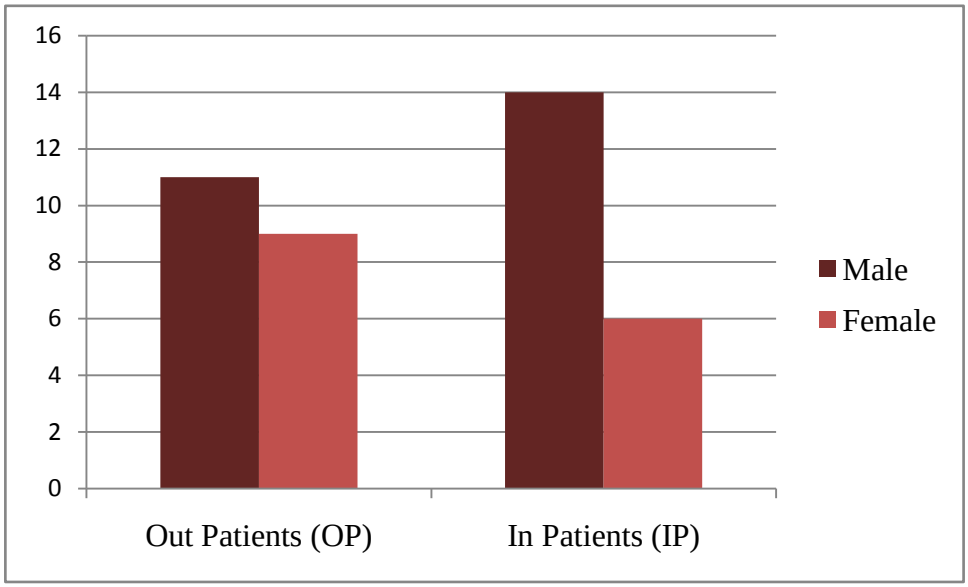
c). Changes in Body Mass Index

1. DISTRIBUTION OF GENDER

TABLE-1 DISTRIBUTION OF GENDER

	Sex	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Male	11	55%	14	70%
2.	Female	09	45%	06	30%
Total		**20**	**100%**	**20**	**100%**

FIGURE-1 DISTRIBUTION OF GENDER



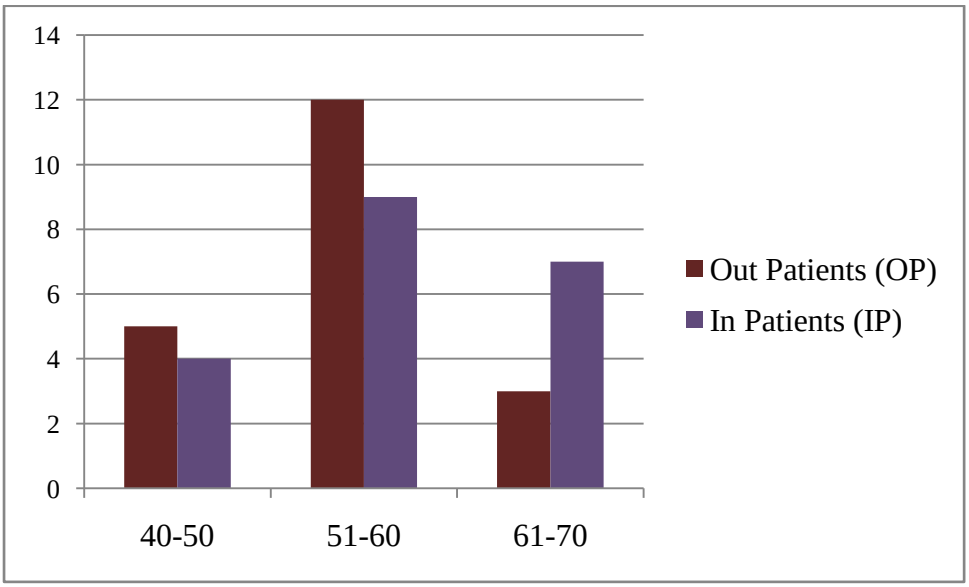
In 40 patients included in the study Akkini Selathumam is not equally distributed in both sexes. Table shows among 20 OP patients 45% were female and 55% were male, among 20 IP patients 70% were male 30% were female.

2. DISTRIBUTION OF AGE

TABLE-2DISTRIBUTION OF AGE

Sl. No.	Age group (In years)	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	40-50	05	25%	04	20%
2.	51-60	12	60%	09	45%
3.	61-70	03	15%	07	35%
Total		**20**	**100%**	**20**	**100%**

FIGURE-2DISTRIBUTION OF AGE



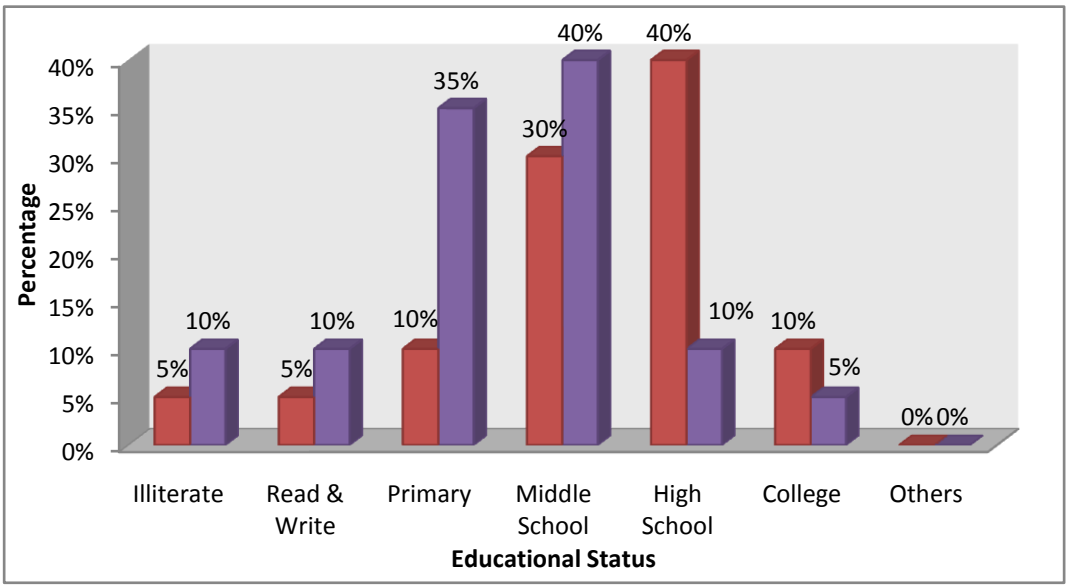
The highest incidence was in Akkini Selathumam. Among the 20 OP and 20 IP patients observed 60% and 45% were affected in age group of 51-60 years, age group of 61-70 closely followed and given among 20 patients in OP 15% were affected and among 20 patients in IP 35% were affected.

3. .DISTRIBUTION OF EDUCATIONAL STATUS

TABLE-3 DISTRIBUTION OF EDUCATIONAL STATUS

Sl. No.	Educational Status	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Illiterate	1	5%	2	10%
2.	Read & Write	1	5%	2	10%
3.	Primary	2	10%	5	35%
4.	Middle School	6	30%	8	40%
5.	High School	8	40%	2	10%
6.	College	2	10%	1	5%
7.	Others	-	-	-	-
Total		**20**	**100%**	**20**	**100%**

FIGURE-3 DISTRIBUTION OF EDUCATIONAL STATUS



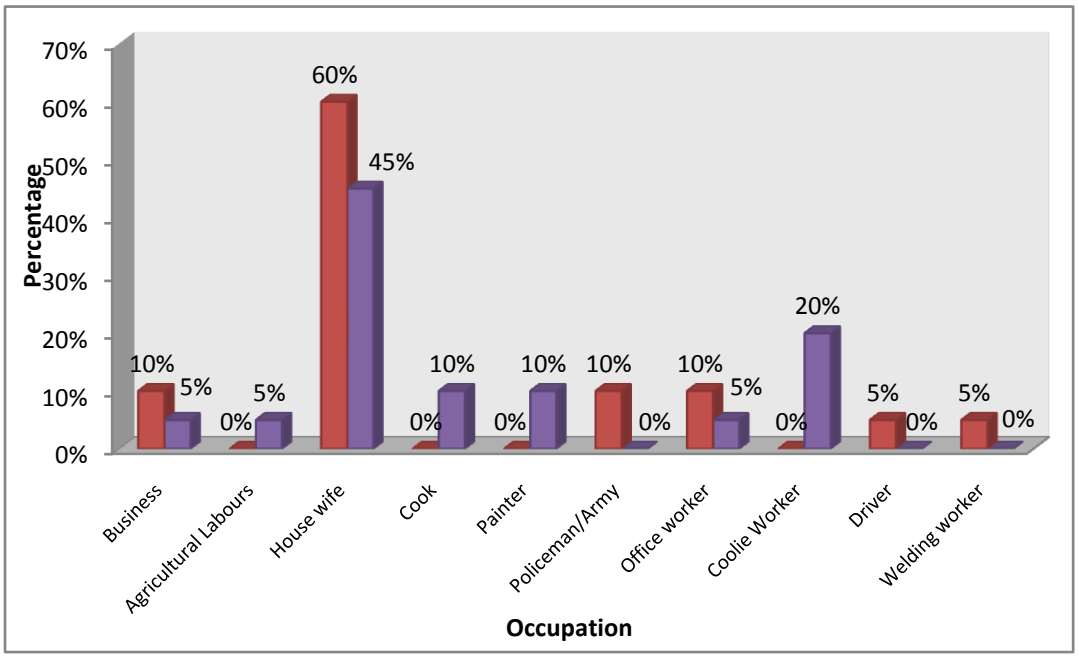
From the above table 3 and Fig 3. it is observed that highest incidence of Akkini Selathumam among 20 out patients was attended high school with 40% and among 20 in patients was attended middle school with 40%.

4. DISTRIBUTION OF OCCUPATION

TABLE-4 DISTRIBUTION OF OCCUPATION

Sl. No.	Occupation	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Business	2	10%	1	5%
2.	Agricultural Labours	-	-	1	5%
3.	House wife	12	60%	9	45%
4.	Cook	-	-	2	10%
5.	Painter	-	-	2	10%
6.	Policeman/Army	2	10%	-	-
7.	Office worker	2	10%	1	5%
8.	Coolie Worker	-	-	4	20%
9.	Driver	1	5%	-	-
10.	Welding worker	1	5%	-	-
Total		**20**	**100%**	**20**	**100%**

FIGURE-4DISTRIBUTIONOFOCCUPATION



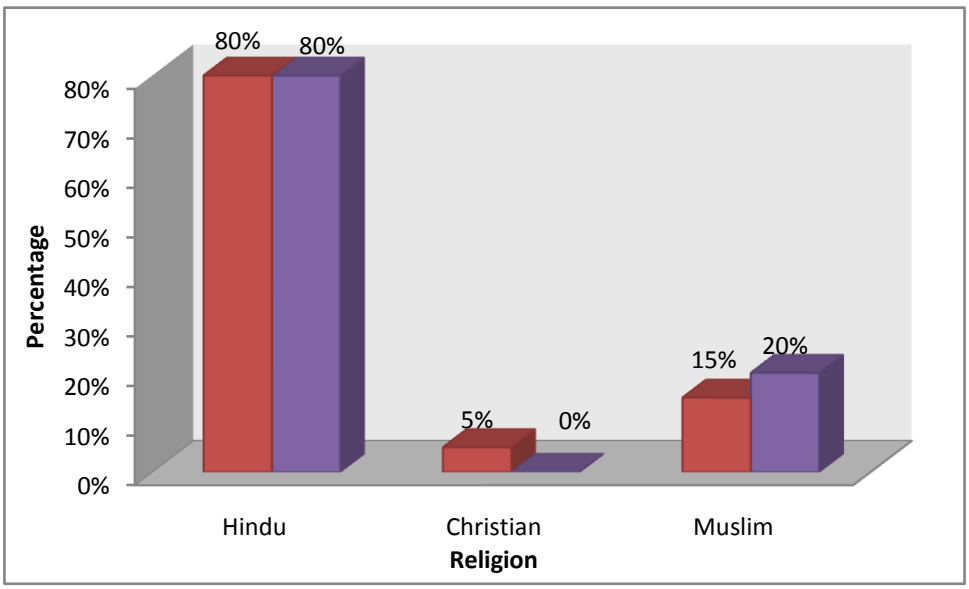
The above Table no 4 & Fig 4. Showed the total Population employed in various occupations have observed the highest incidence of Akkini Selathumam.The result found 60% OP 45% IP patients were house wives.

5. DISTRIBUTION OF RELIGION

TABLE-5 DISTRIBUTION OF RELIGION

Sl. No.	Religion	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Hindu	16	80%	16	80%
2.	Christian	1	5%	-	-
3.	Muslim	3	15%	4	20%
Total		**20**	**100%**	**20**	**100%**

FIGURE-5 DISTRIBUTION OF RELIGION



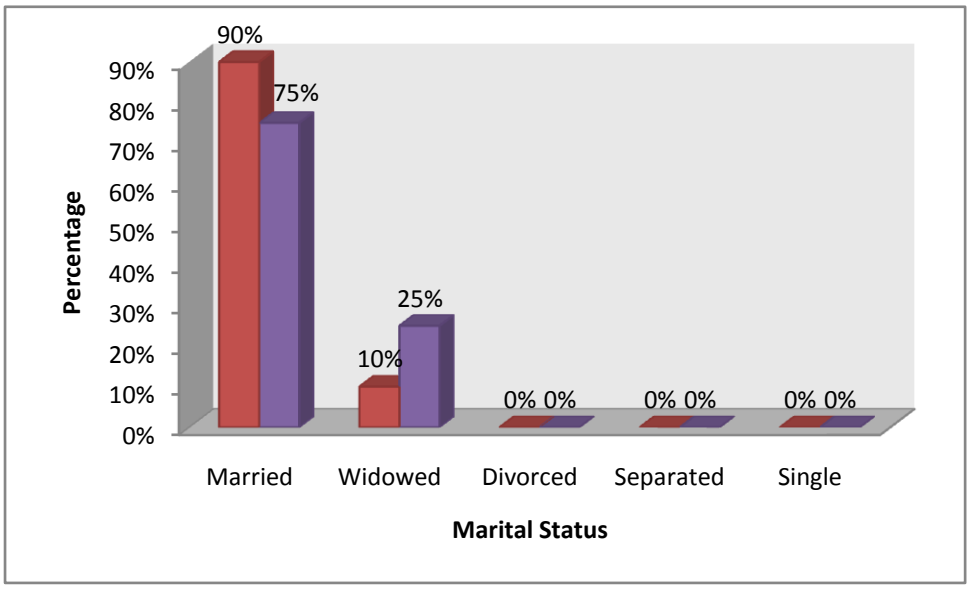
From the table 5 and Fig no 5 reveals that 80% were Hindus, 15% were Muslims and 5% were Christians.

6. DISTRIBUTION OF MARITAL STATUS

TABLE-6 DISTRIBUTION OF MARITAL STATUS

Sl. No.	Marital Status	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Married	18	90%	15	75%
2.	Widowed	2	10%	5	25%
3.	Divorced	-	-	-	-
4.	Separated	-	-	-	-
5.	Single	-	-	-	-
Total		**20**	**100%**	**20**	**100%**

FIGURE-6 DISTRIBUTION OF MARITAL STATUS



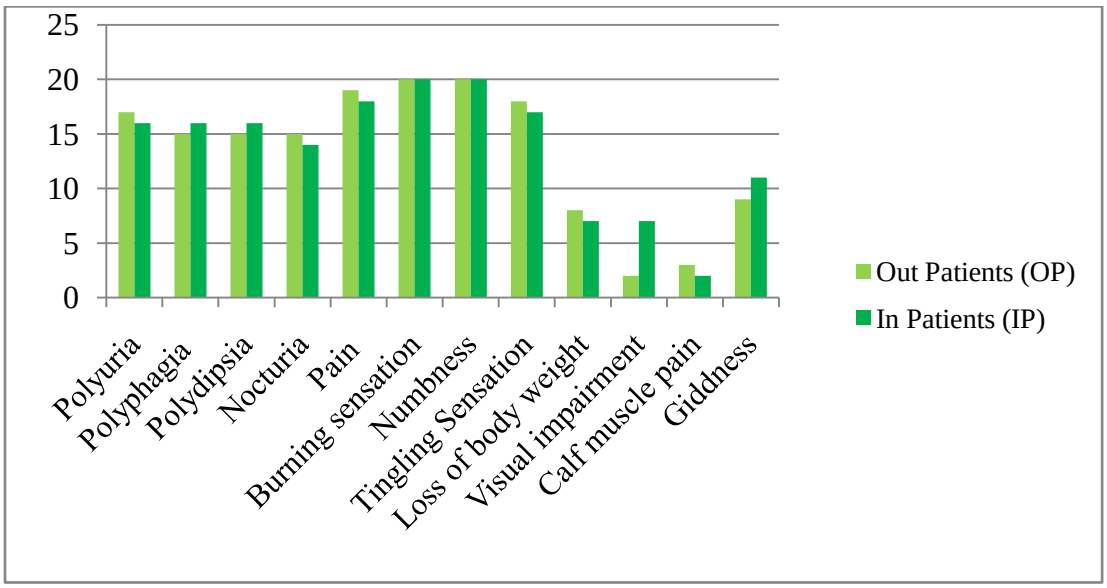
The Table and Fig no 6 showed.the highest incidence is 90% in married and 10% in Widowed at OPD .The IPD was 75% and 25% respectively.

7. DISTRIBUTION OF CLINICAL MANIFESTATION

TABLE-7DISTRIBUTION OF CLINICAL MANIFESTATION

Sl. No.	Clinical Manifestation	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Polyuria	17	85%	16	80%
2.	Polyphagia	15	75%	16	80%
3.	Polydipsia	15	75%	16	80%
4.	Nocturia	15	75%	14	70%
5.	Pain	19	95%	18	90%
6.	Burning sensation	20	100%	20	100%
7.	Numbness	20	100%	20	100%
8.	Tingling Sensation	18	90%	17	75%
9.	Loss of body weight	8	40%	7	35%
10.	Visual impairment	2	10%	7	35%
11.	Calf muscle pain	3	15%	2	10%
12.	Giddness	9	45%	11	55%

FIGURE-7 DISTRIBUTION OF CLINICAL MANIFESTATION



From the above table 7 showed that 20 Out patients, 85% of cases had polyuria and 75%,polydipsia and 75% had Nocturia. 95% had pain, 100% Burning sensation, 100% had Numbness, 90% had Tingling Sensation, Loss of body weight 40% . 10% had visual impairment, 15% had calf muscle pain. 100% clinical symptoms were presentd in burning pain and numbness.

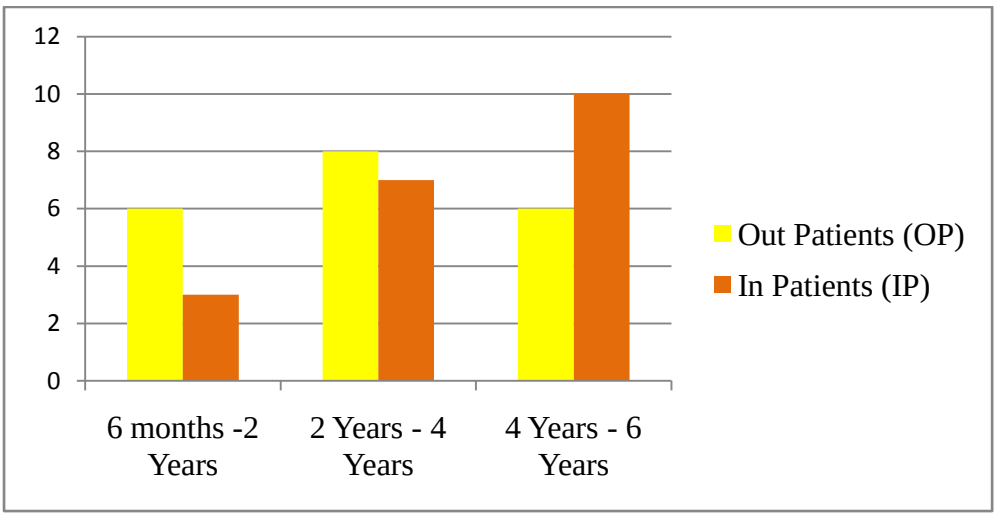
The 80% of in patients cases had polyuria polydipsia and polyphagia, and 100% had Burning sensation. 90% had pain, 70% nocturia, 100% had Numbness, 75% had Tingling Sensation, 40% had loss of body weight, 35% had visual impairment, 10% had calf muscle pain and 55% had Giddiness.

8. **DISTRIBUTION OF DURATION OF ILLNESS**

TABLE-8 DISTRIBUTION OF DURATION OF ILLNESS

Sl. No.	Duration of illness	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
3.	6 months -2 Years	6	30%	3	15%
4.	2 Years - 4 Years	08	40%	07	35%
5.	4 Years - 6 Years	6	30%	10	50%
Total		**20**	**100%**	**20**	**100%**

FIGURE-8 DISTRIBUTION OF DURATION OF ILLNESS



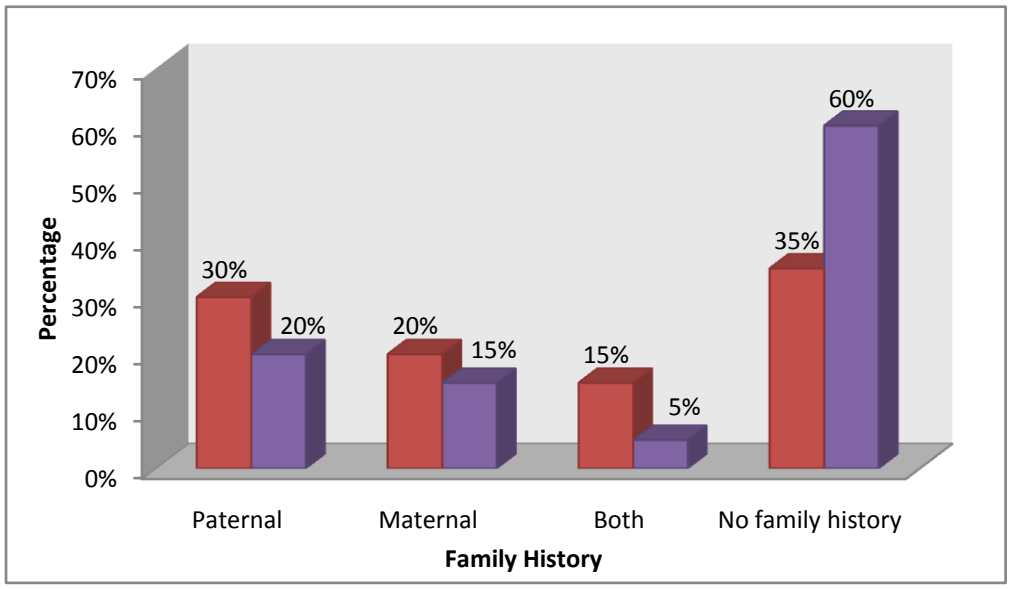
According to Table &Fig 8 showed that Akkini Selathumam was greatly noticed in the 40 patients were suffering with in madhumegam was incidence about more than 2 years to 4 years. In IPD the percentage of madhumegam was 40% in OPD and 35% in IPD.

9. DISTRIBUTION OF FAMILY HISTORY

TABLE-9 DISTRIBUTION OF FAMILY HISTORY

Sl. No.	Family History	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Paternal	6	30%	4	20%
2.	Maternal	4	20%	3	15%
3.	Both	3	15%	1	5%
4.	No family history	7	35%	12	60%
Total		**20**	**100%**	**20**	**100%**

FIGURE-9 DISTRIBUTION OF FAMILY HISTORY



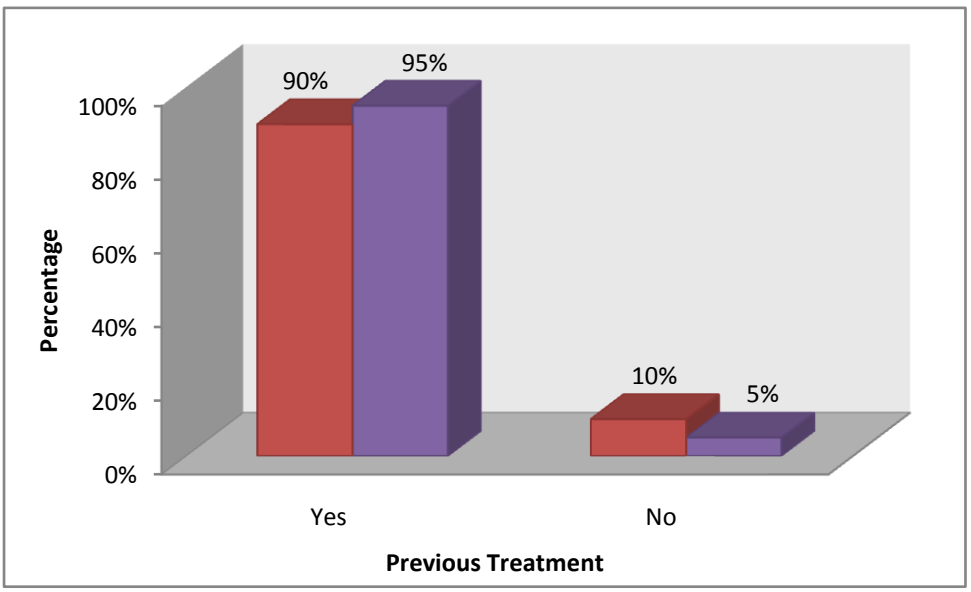
The table & Fig no 9 showed the incidence of Akkini selathumam in family history were presence in OP 30% had paternal history, 20% had maternal history, 15% had both and 35% had no family history. Among IP 20% of IP patient had paternal history, 15% had maternal history and 5% had both history.

10. **DISTRIBUTION OF HISTORY OF PREVIOUS TREATMENT OF MADHUMEGAM**

TABLE-10 DISTRIBUTION OF PREVIOUS TREATMENT

Sl. No.	Previous Treatment	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Yes	18	90%	19	95%
2.	No	2	10%	1	5%
Total		**20**	**100%**	**20**	**100%**

FIGURE-10 DISTRIBUTION OF PREVIOUS TREATMENT



The Table and Fig shows that 90% of OP and 95% of IP patients had a history of previous treatment for Madhumegam.

11. DISTRIBUTION OF PERSONAL HISTORY

TABLE-11 DISTRIBUTION OF PERSONAL HISTORY

Sl. No.	Personal History	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	**DIET**				
	Vegetarian	6	30%	7	35%
	Non Veg	-	-	-	-
	Mixed Diet	14	70%	13	65%

FIGURE-11 DISTRIBUTION OF PERSONAL HISTORY

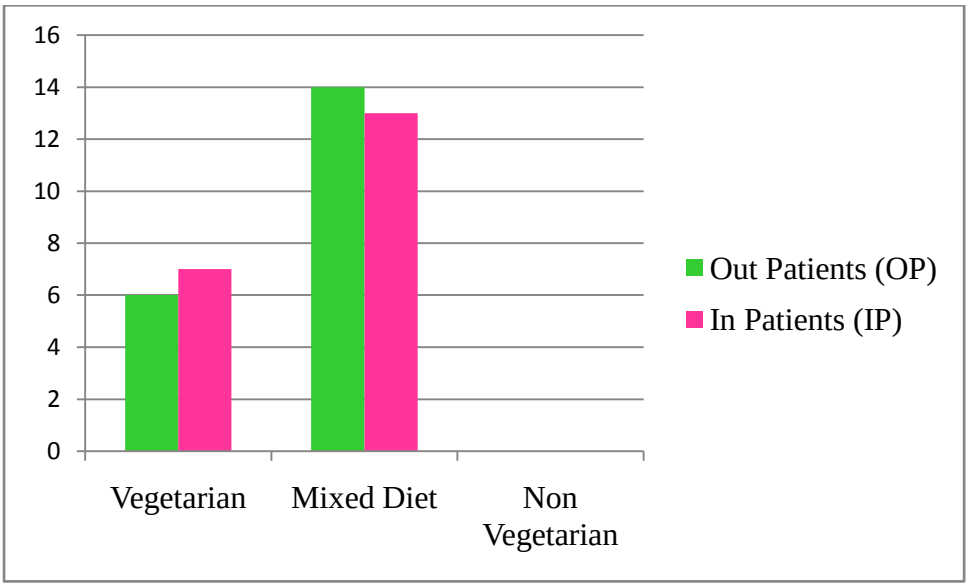


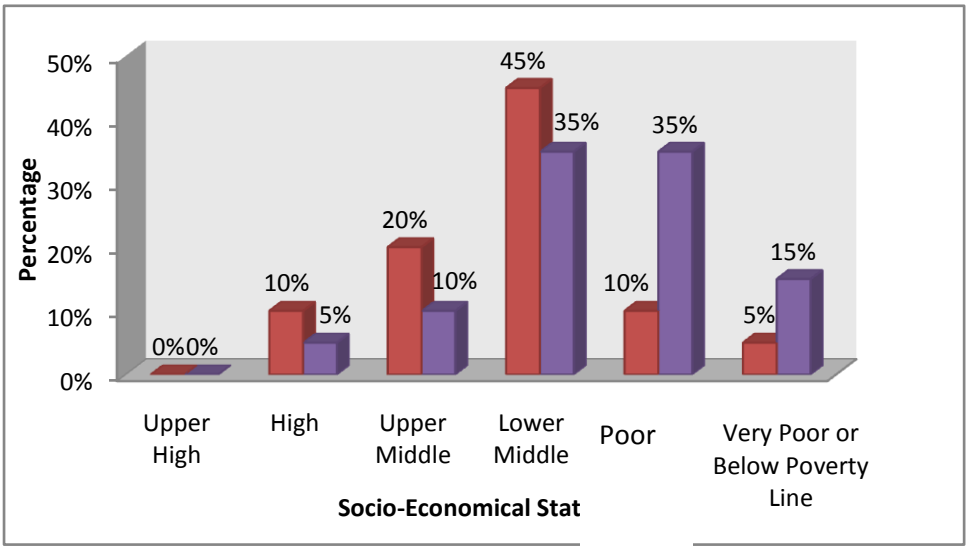
Table and Fig no 11.The Akkini selathumam was reported the 95% of OP and 90% of IP patients have mixed diet.

12. DISTRIBUTION OF SOCIO-ECONOMICAL STATUS

TABLE-12 DISTRIBUTION OF SOCIO-ECONOMICAL STATUS

Sl. No.	Socio-Economical Status	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Upper High	-	-	-	-
2.	High	3	10%	1	5%
3.	Upper Middle	5	20%	2	10%
4.	Lower Middle	9	45%	7	35%
5.	Poor	2	10%	7	35%
6.	Very Poor or Below Poverty Line	1	5%	3	15%
Total		**20**	**100%**	**20**	**100%**

FIGURE-12 DISTRIBUTION OF SOCIO-ECONOMICAL STATUS



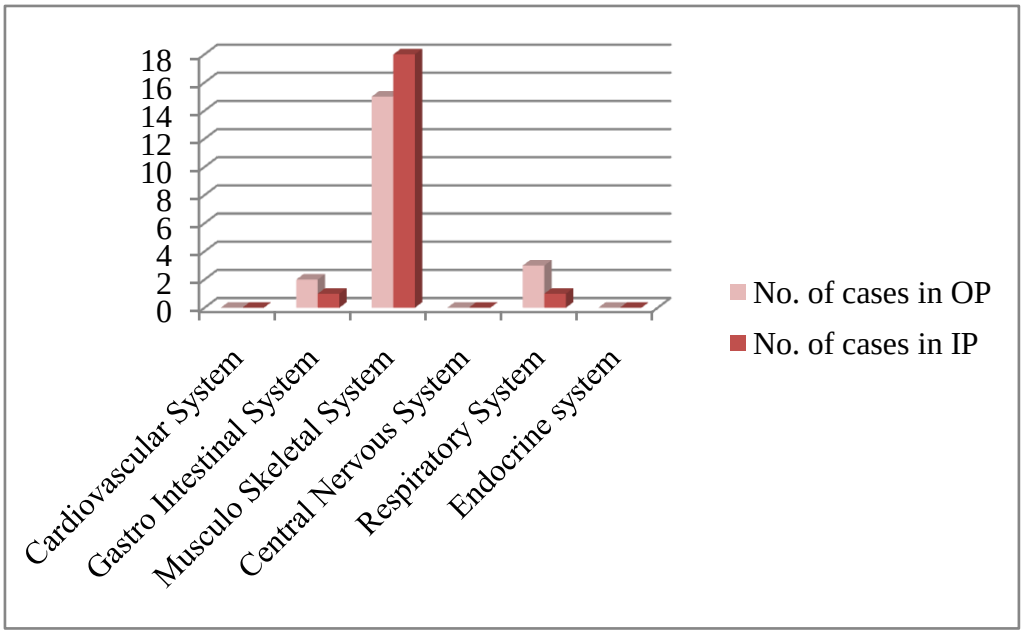
Among the 40 patients of the study group there is a marked percentage of the disease of the population of lower middle socio economic status in relative percentages of 45% were in OP and 35% were in IP.The heighest incidence was lower middle class peoples (Table no 12).

13. **DISTRIBUTION OF OTHER SYSTEM INVOLVEMENT**

TABLE-13 DISTRIBUTION OF OTHER SYSTEM INVOLVEMENT

Sl. No.	System Involvement	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Cardiovascular System	-	-	-	-
2.	Gastro Intestinal System	2	10%	1	5%
3.	Musculo Skeletal System	15	75%	18	90%
4.	Central Nervous System	-	-	-	-
5.	Respiratory System	3	10%	1	5%
6.	Endocrine system	-	-	-	-
Total		**20**	**100%**	**20**	**100%**

FIGURE-13 DISTRIBUTION OF OTHER SYSTEM INVOLVEMENT



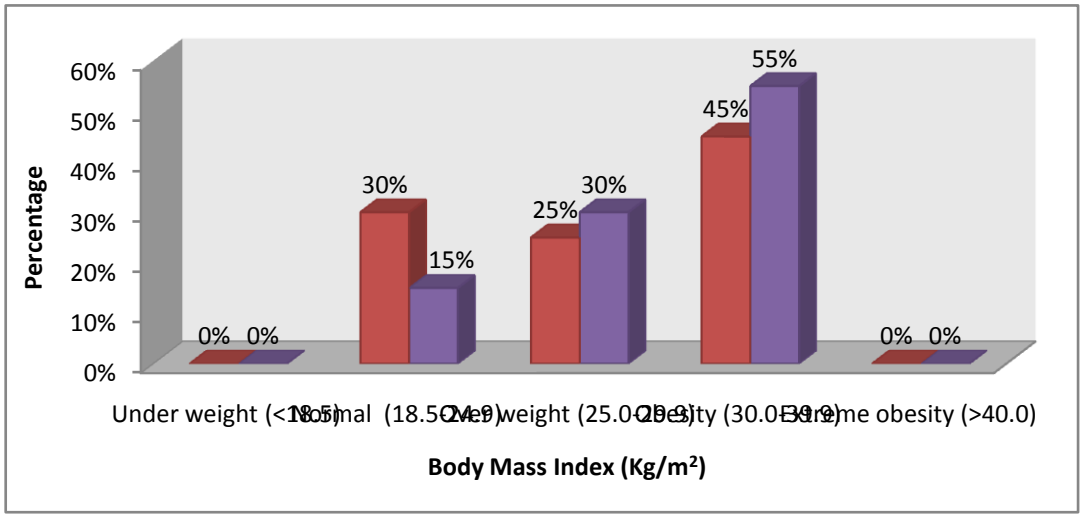
The table & Fig no 13 showed the System involvement of Akkini selathumam were presence in OP 10% had GIT System, 75% had Musculo Skeletal System, 15% had both and 35% had no family history. IP 90% of IP patient had Musculo Skeletal System, 15% had Respiratory System and 5% had both system involved.

14. DISTRIBUTION OF BODY MASS INDEX

TABLE-14 BODY MASS INDEX

Sl. No.	Body Mass Index (Kg/m^2)	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Under weight (<18.5)	-	-	-	-
2.	Normal (18.5-24.9)	6	30%	3	15%
3.	Over weight (25.0-29.9)	5	25%	6	30%
4.	Obesity (30.0-39.9)	9	45%	11	55%
5.	Extreme obesity (>40.0)	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-14 BODY MASS INDEX



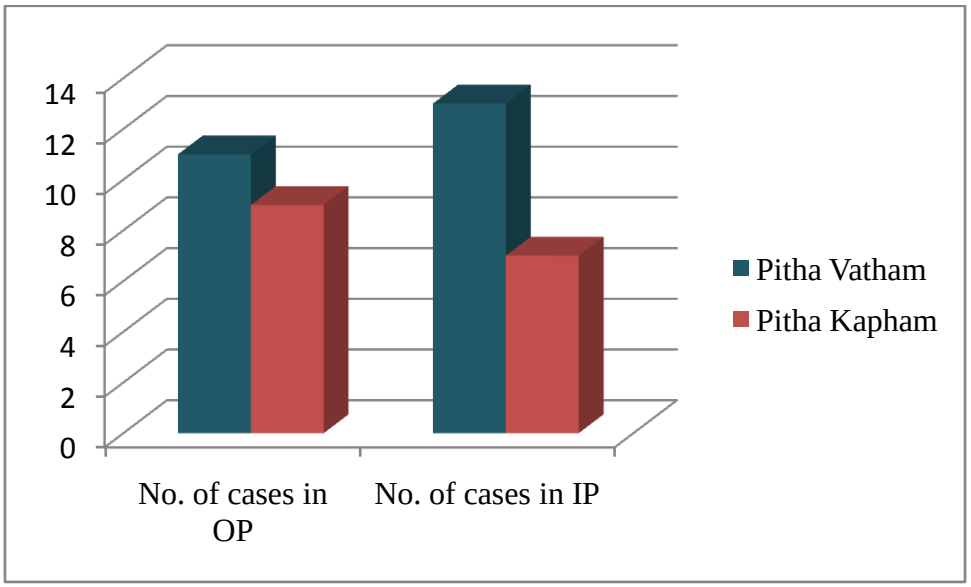
The table and Fig No 14 shows that the body mass index the 40 patients of the study group patients had normal BMI 30% in OP and 15% in IPD. 45% in OP and 55% in IP patients had obesity (Class-I &II). 25% in OP 30% in IP patients having (over weight) in Akkini Selathumam.

15. DISTRIBUTION OF CONSTITUTION OF BODY

TABLE-15 DISTRIBUTION OF CONSTITUTION OF BODY

Sl. No.	Constitution of body	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Pitha Vatham	11	55%	13	65%
2.	Pitha Kapham	09	45%	07	35%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-15 DISTRIBUTION OF CONSTITUTION OF BODY



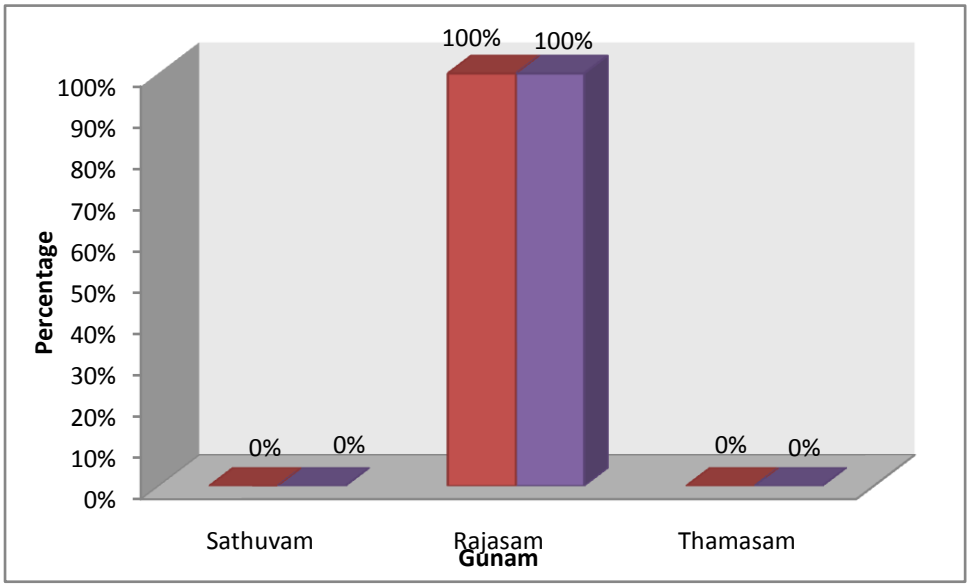
From the above table 14 and Fig no 14 was reveals that Akkini Selathumam have 20 Out patients were Pitha Vatha Thegi with 55%, Pitha kapa45%, In IP patients Pitha Vatha Thegi with 65%, Pitha Kapa Thegi with 35% were observed from this study.

16. DISTRIBUTION OF GUNAM

TABLE-16 DISTRIBUTION OF GUNAM

Sl. No.	Gunam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Sathuvam	-	-	-	-
2.	Rajasam	20	100%	20	100%
3.	Thamasam	-	-	-	-
Total		**20**	**100%**	**20**	**100%**

FIGURE-16 DISTRIBUTION OF GUNAM



From the above Table and Fig 16 is showed that highest incidence of Akkini selathumam among Out patients and In patients with cent percent belongs to Rajo Gunam.

17. DISTRIBUTION OF KAALAM

TABLE-17 DISTRIBUTION OF KAALAM

Sl. No.	Kaalam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Vatham (1-33 Years)	-	-	-	-
2.	Pitham (34-66 Years)	15	75%	17	85%
3.	Kapham (67- 100 Years)	5	25%	3	15%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-17 DISTRIBUTION OF KAALAM

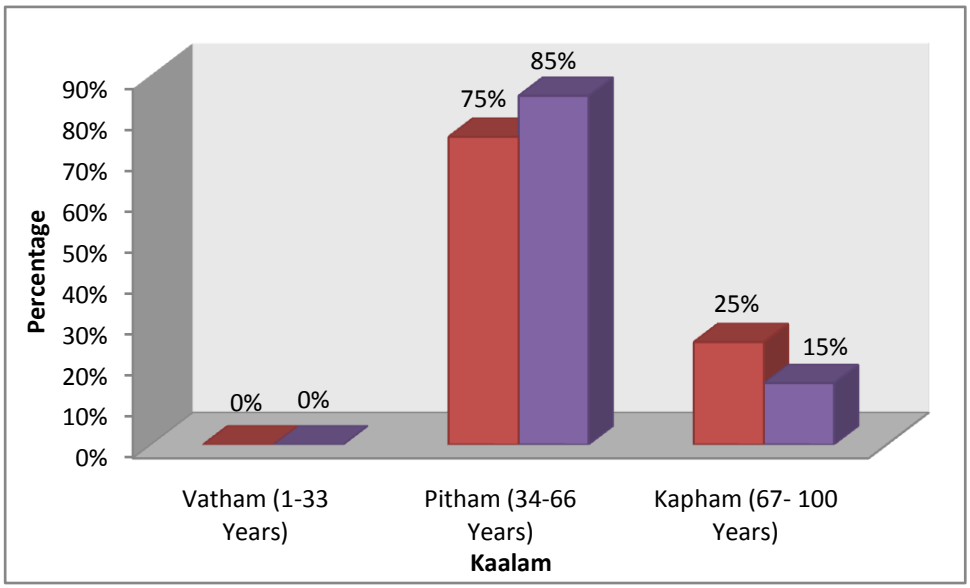


Table and Fig no 17 is observed from this study, the highest incidence of Akkini selathumam among 20 Out patients is in Pitha Kaalam with 75%, Kapha Kaalam with 25%. Among 20% In patients, is also Pitha Kaalam with 85%, Kapha Kaalam with 15%.

18. **DISTRIBUTION OF PARUVA KAALAM**

TABLE-18 DISTRIBUTION OF PARUVA KAALAM

Sl. No.	Paruva Kaalam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Elavenil Kaalam	-	-	-	-
2.	Muthuvenil Kaalam	-	-	-	-
3.	Kaar Kaalam	-	-	1	5%
4.	Koothir Kaalam	5	25%	-	-
5.	Munpani Kaalam	15	75%	2	10%
6.	Pinpani Kaalam	-	-	17	85%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-18 DISTRIBUTION OF PARUVA KAALAM

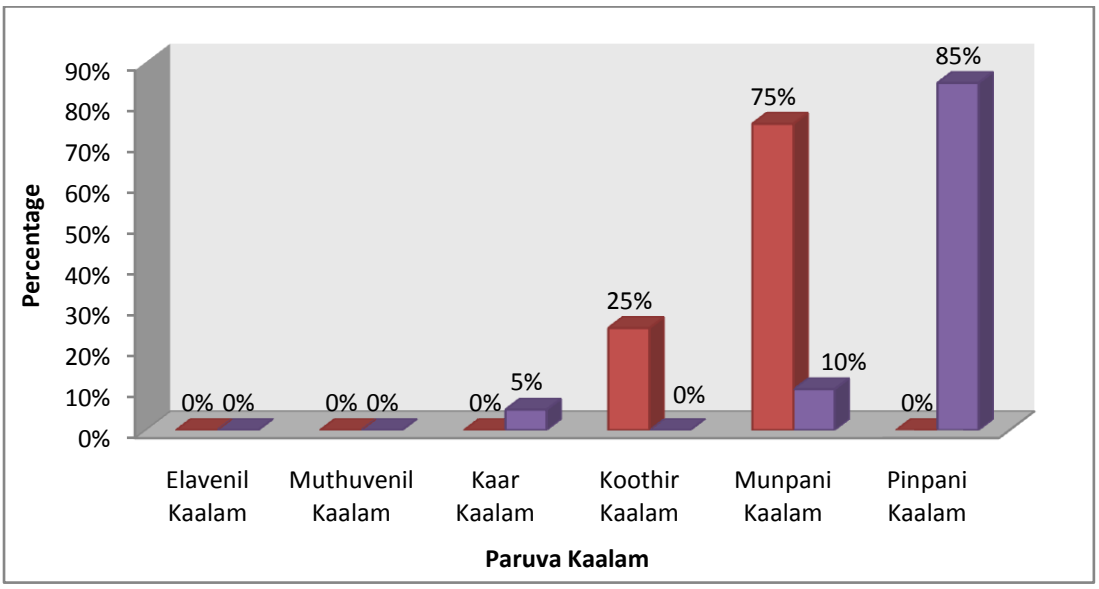


Table and Fig No 18 is the highest incidence of Akkini Selathumam among 20 Out patients highest incidence of the disease is in Munpani Kaalam 75% and among 20 In patient were in Pinpani Kaalam 85%.

19. DISTRIBUTION OF THINAI

TABLE-19 DISTRIBUTION OF THINAI

Sl. No.	Thinai	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Kurinji	1	5%	1	5%
2.	Mullai	-	-	-	-
3.	Marutham	18	90%	19	95%
4.	Neithal	1	5%	-	-
5.	Paalai	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-19 DISTRIBUTION OF THINAI

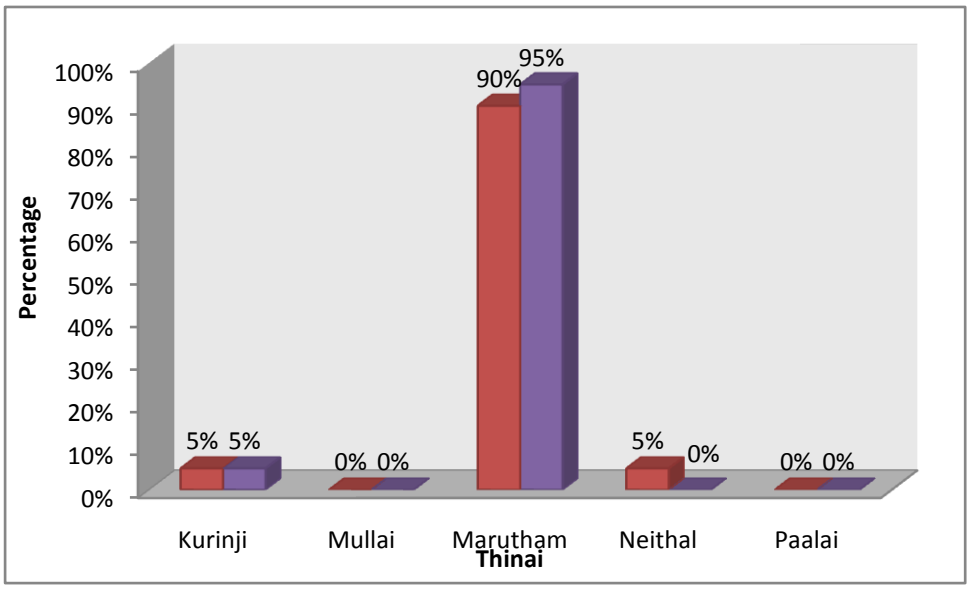


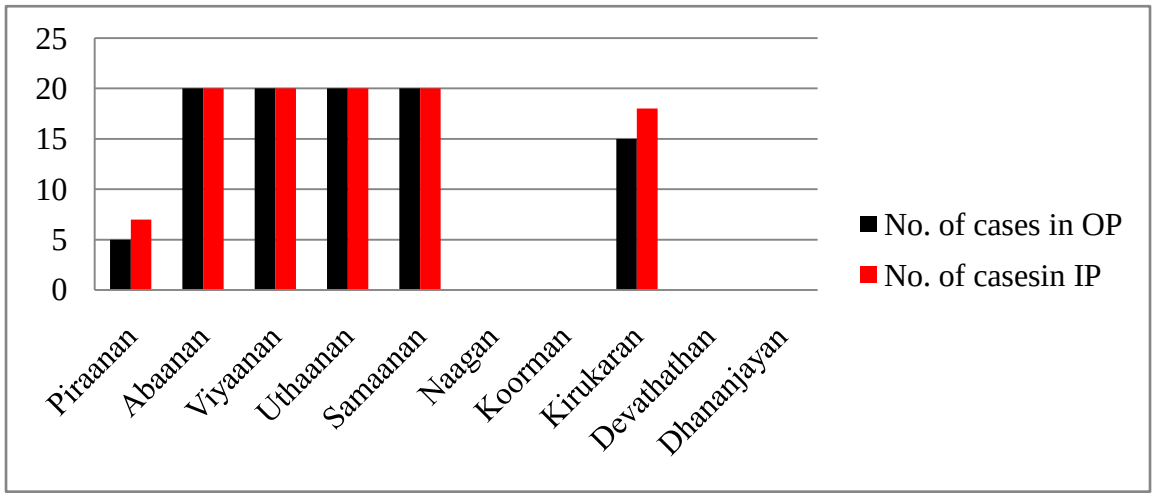
Table and Fig 19 showed that the highest incidence of Madhumega Avathaigal-I among 20 Out patients were in the Marutham land is 90% and among In Patients also affected in Marutham land with 95%.

20. DISTRIBUTION OF MUKKUTRAM
(a). DERANGEMENT OF VATHAM

TABLE-20 (a) DERANGEMENT OF VATHAM

Sl. No.	Derangement of Vatham	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Piraanan	05	25%	7	35%
2.	Abaanan	20	100%	20	100%
3.	Viyaanan	20	100%	20	100%
4.	Uthaanan	20	100%	20	100%
5.	Samaanan	20	100%	20	100%
6.	Naagan	-	-	-	-
7.	Koorman	-	-	-	-
8.	Kirukaran	15	75%	18	90%
9.	Devathathan	-	-	-	-
10.	Dhananjayan	-	-	-	-

FIGURE-20 (a) CONDITION OF VATHAM



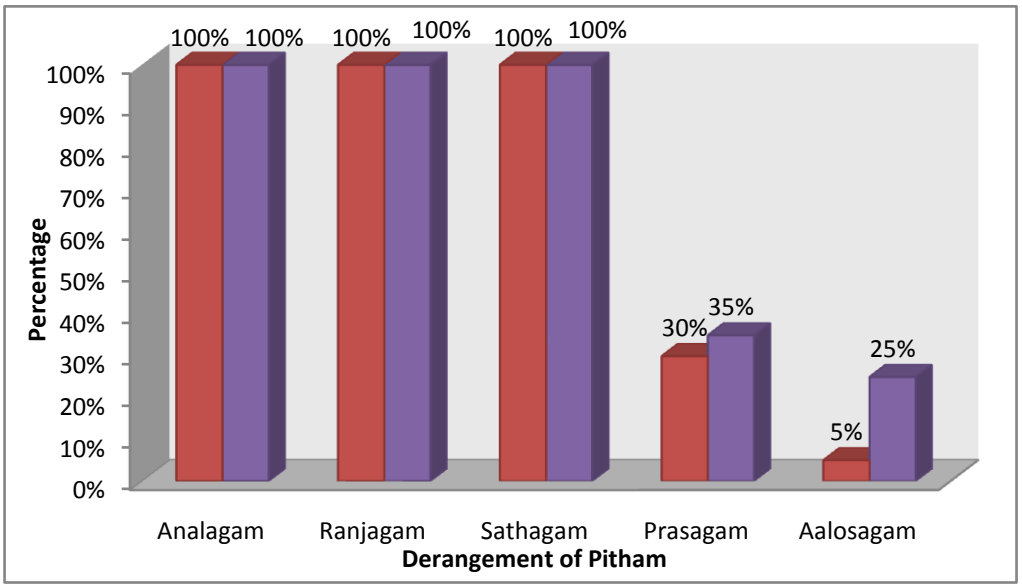
The table and Figure no 20 (a) showed that among 20 out patients 100% were affected in Viyaanan, Samaanan, Uthaanan and Abaanan; 75% were affected in kirukaran. Among 20 In patients 100% were affected in Viyaanan, Samaanan, Uthaanan and Abaanan. 90% were affected in kirukaran.

20 (b) DERANGEMENT OF PITHAM

TABLE-20 (b) DERANGEMENT OF PITHAM

Sl. No.	Derangement of Pitham	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Analagam	20	100%	20	100%
2.	Ranjagam	20	100%	20	100%
3.	Sathagam	20	100%	20	100%
4.	Prasagam	6	30%	7	35%
5.	Aalosagam	1	5%	5	25%

FIGURE-20 (b) CONDITION OF PITHAM



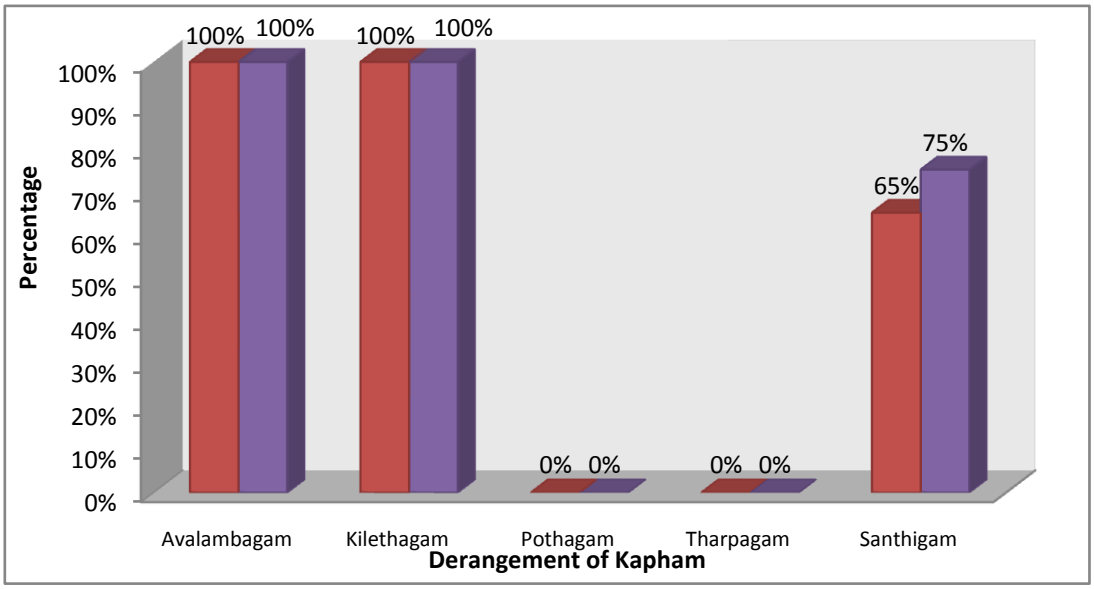
The table and Fig no 20(b) is reveal that among 20 Out patients 100% were affected in Analagam, Ranjagam and Sathagam pitham; 30% were affected in Prasagam; 5% were affected in Aalosagam; Among 20 In patients 100% were affected in Analagam, Ranjagam and Sathagam; 35% were affected in Prasagam; 25% were affected in Aalosagam pitham.

20(c) DERANGEMENT OF KAPHAM

TABLE-20 (c) DERANGEMENT OF KAPHAM

Sl. No.	Derangement of Kapham	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Avalambagam	20	100%	20	100%
2.	Kilethagam	20	100%	20	100%
3.	Pothagam	-	-	-	-
4.	Tharpagam	-	-	-	-
5.	Santhigam	13	65%	15	75%

FIGURE-20 (c) DERANGEMENT OF KAPHAM



According Table and Fig no 20 (c).the Akkini selathumam was reported the 20 Out patients 100% were affected in Avalambagam and Kilethagam; 65% were affected in Santhigam pitham. Among 20 In patients100% were affected in Avalambagam and Kilethagam 75% were affected in Santhigam.

21. DISTRIBUTION OF INVOLVEMENT OF UDAL THATHUKKAL

TABLE-21 INVOLVEMENT OF UDAL THATHUKKAL

Sl. No.	Involvement of Udal Thathukkal	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Saaram	20	100%	20	100%
2.	Senneer	20	100%	20	100%
3.	Oon	13	65%	15	75%
4.	Kozhuppu	16	80%	17	85%
5.	Enbu	4	20%	5	25%
6.	Moolai	-	-	-	-
7.	Sukkilam / Suronitham	1	5%	-	-

FIGURE-21 INVOLVEMENT OF UDAL THATHUKKAL

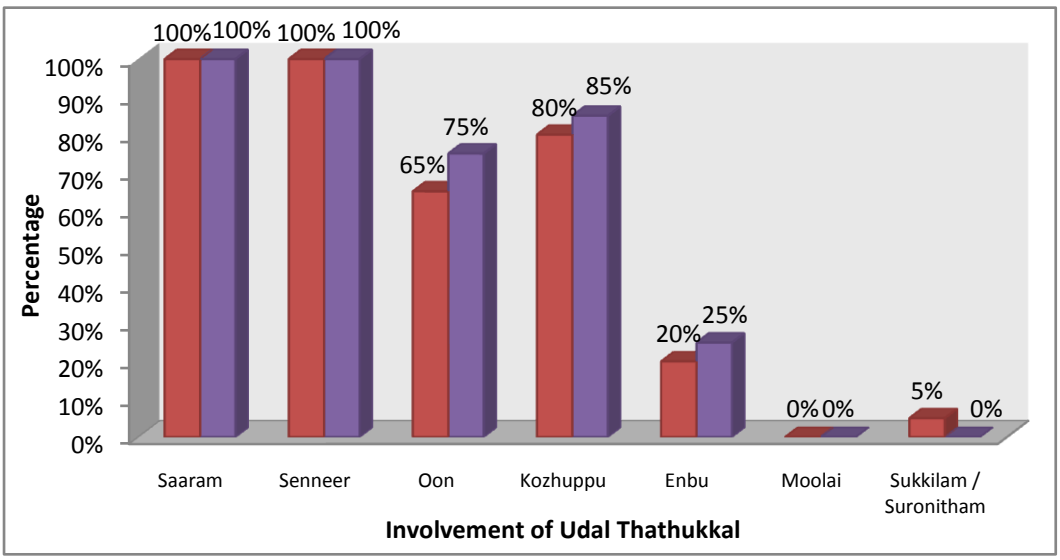


Table and Figure No 21 is showed that among 20 Out patients and 20 In patients 100% were affected in Saaram and Senneer, 65% were affected in Kozhuppu and 20% were affected in Enbu. Among 20 Out patients 100% were affected in Saaram and Senneer, 75% were affected in Kozhuppu and 20% were affected in Enbu thathu.

22. DISTRIBUTION OF KANMENTHIRIYAM

TABLE-22 DISTRIBUTION OF KANMENTHIRIYAM

Sl. No.	Kanmenthiriyam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Kai	05	25%	8	40%
2.	Kaal	20	100%	20	100%
3.	Vai	-	-	-	-
4.	Eruvai	12	60%	14	70%
5.	Karuvai	-	-	-	-

FIGURE-22 DISTRIBUTION OF KANMENTHIRIYAM

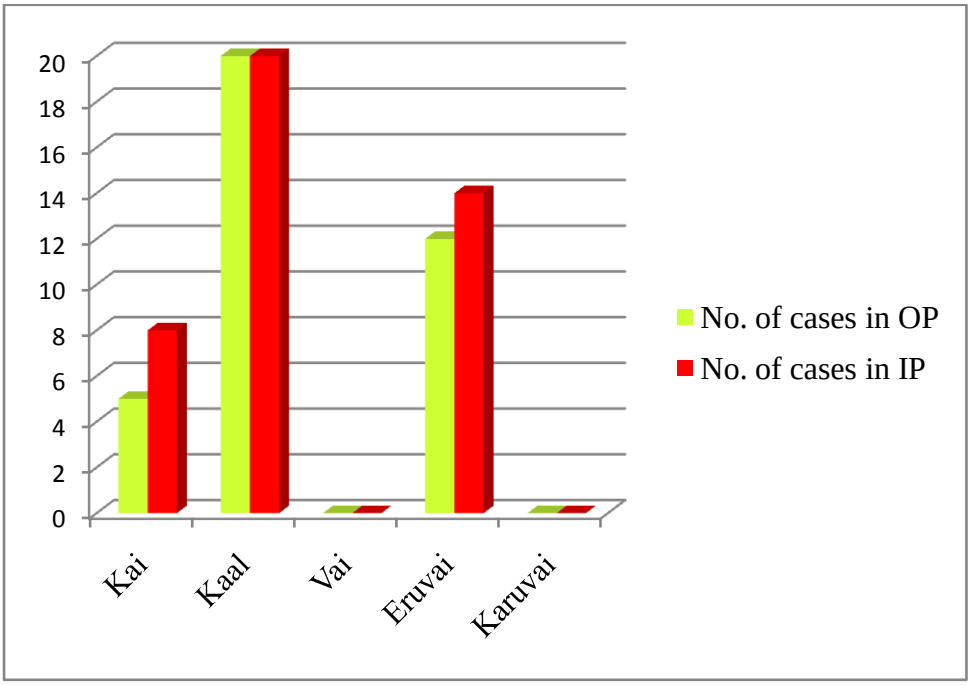


Figure and Table.22 it is observed that among 20 Out patients, 100% were affected in Kaal, 65% in Kai and 60% in Eruvai. Among 20 In patients 100% were affected in Kaal and 70% in Eruvai was affected.

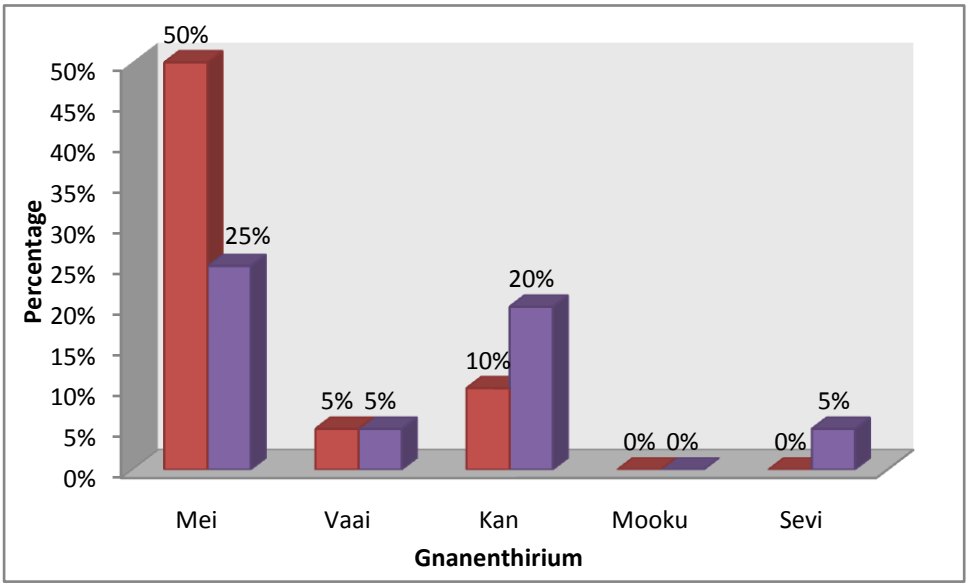
23. DISTRIBUTION OF IMPORIGAL(GNANENDRIUM)

.

TABLE-23 DISTRIBUTION OF IMPORIGAL (GNANENTHIRIUM)

Sl. No.	Gnanenthirium	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Mei	10	50%	5	25%
2.	Vaai	1	5%	1	5%
3.	Kan	2	10%	4	20%
4.	Mooku	-	-	-	-
5.	Sevi	-	-	1	5%

FIGURE-23 DISTRIBUTION OF IMPORIGAL (GNANENTHIRIUM)



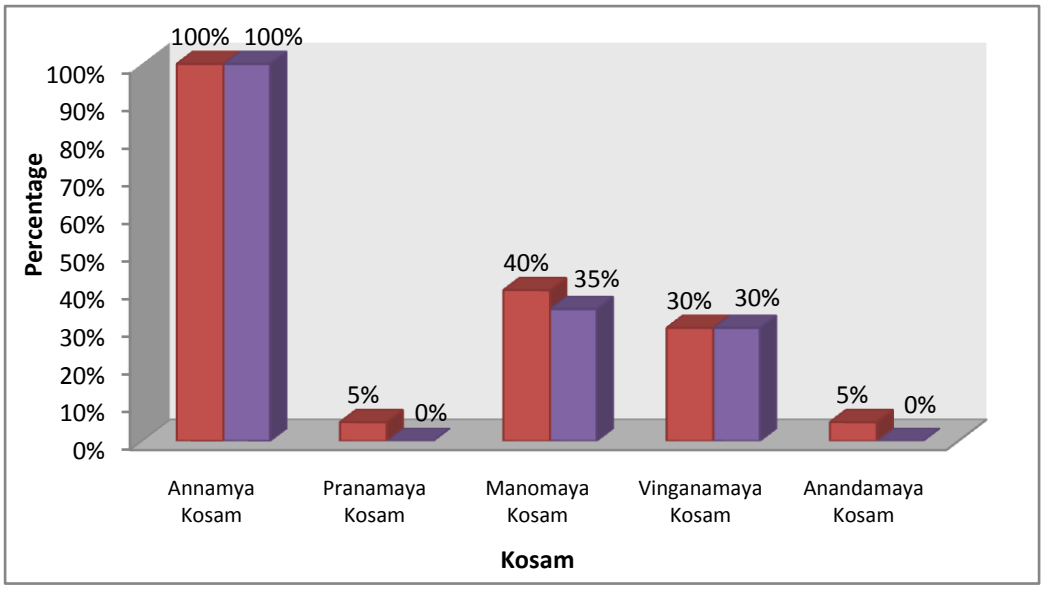
From the above table and Fig no.23 is showed that among 20 Out patients and 20 In patients 50% and 25% were affected in Mei. Among 20 Out patients 10% and 20 In patients 20% were affected in Kan.

24. DISTRIBUTION OF KOSAM

TABLE-24 DISTRIBUTION OF KOSAM

Sl. No.	Kosam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Annamya Kosam	20	100%	20	100%
2.	Pranamaya Kosam	1	5%	-	-
3.	Manomaya Kosam	8	40%	7	35%
4.	Vinganamaya Kosam	6	30%	6	30%
5.	Anandamaya Kosam	1	5%	-	-

FIGURE-24 DISTRIBUTION OF KOSAM



According to Table and Fig 24, is observed that among 20 Out patients 100% were affected in Annamaya Kosam, 40% were affected in Manomaya Kosam and 30% affected in Vinganamaya kosam. Among 20 In patients, 100% were affected in Annamaya Kosam, 35% were affected in Manomaya Kosam and 30% were affected in Vinganamaya Kosam affected.

25. **DISTRIBUTION OF CONDITIONS OF ENVAGAI THERVUGAL**

TABLE-25 DISTRIBUTION OF CONDITIONS OF ENVAGAI THERVUGAL

Sl. No.	Conditions of Envagai Thervugal	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Naadi (Thontha Naadi)				
	3). Pitha Vatham	11	55%	13	65%
	4). Pitha Kapham	9	45%	7	35%
2.	Sparisam	10	50%	5	25%
3.	Naa	12	60%	15	75%
4.	Niram	6	30%	7	35%
5.	Mozhi	-	-	2	10%
6.	Vizhi	2	10%	4	20%
7.	Malam	6	30%	6	30%
8.	Moothiram	15	75%	14	70%

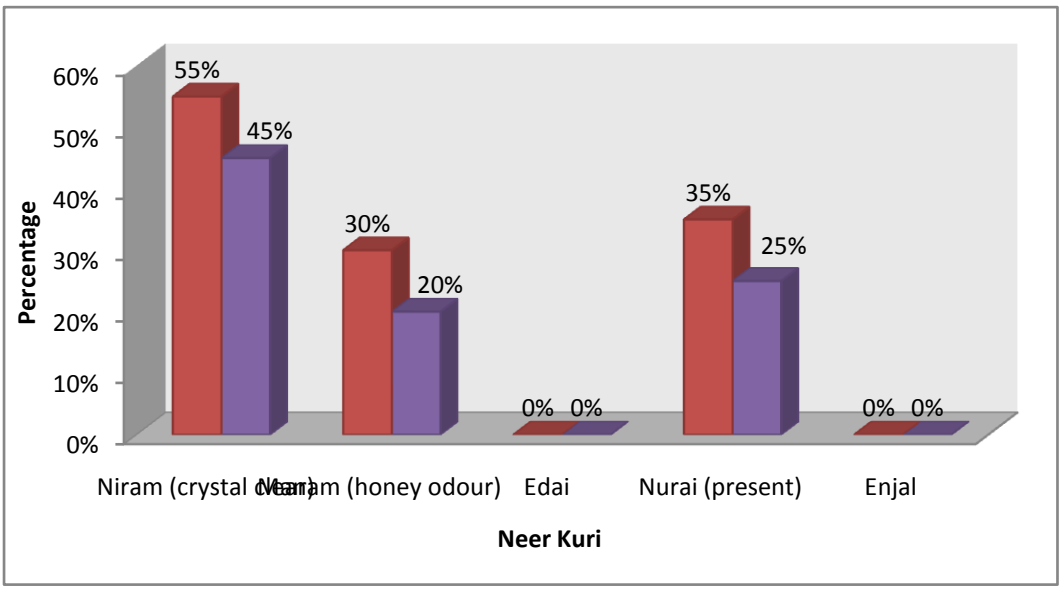
From the table no.25 is observed that among 20 Out patients 55% had Pitha Vatha Naadi, 45% had Pitha Kapha Naadi, 100% were affected in Naa and Moothiram; 50% were affected in Sparisam; 30% were affected in Niram and Malam; 10% were affected in Vizhi. Among 20 In patients 65% had Pitha Vatha Naadi, 35% had Pitha Kapha Naadi; 100% were affected in Naa and Moothiram; 25% were affected in Sparisam;35% were affected in Niram; 30% were affected in Malam; 20% were affected in Vizhi

26. DISTRIBUTION OF NEER KURI

TABLE-26 DISTRIBUTION OF NEER KURI

Sl. No.	Neer Kuri	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Niram (crystal clear)	11	55%	9	45%
2.	Manam (honey odour)	6	30%	4	20%
3.	Edai	-	-	-	-
4.	Nurai (present)	7	35%	5	25%
5.	Enjal	-	-	-	-

FIGURE-26 DISTRIBUTION OF NEER KURI



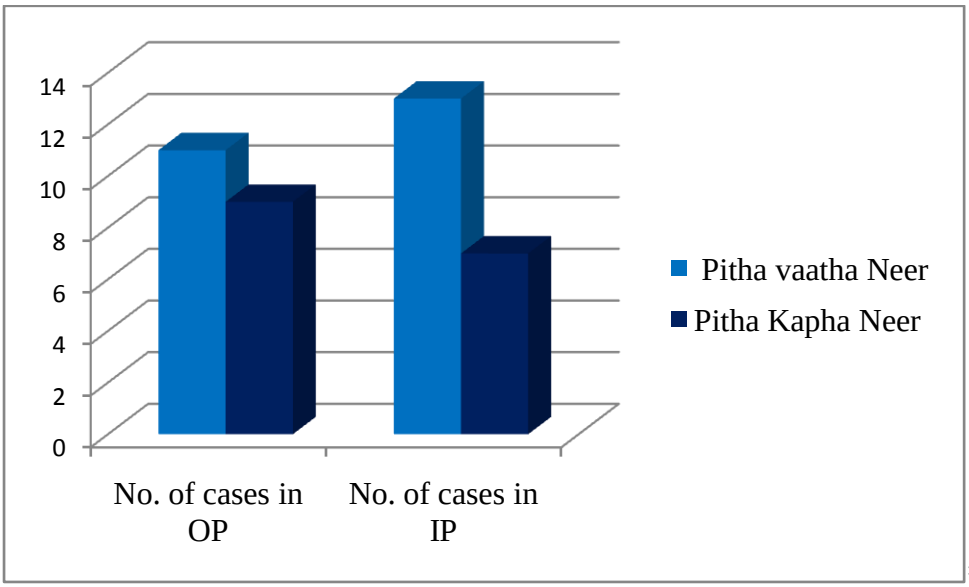
According to table and Fig no 26 showed that, among 20 Out patients 55% were affected in Niram; 30% were affected in manam; 35% were affected in Nurai.Among 20 In patients 45% were affected in Niram; 20% were affected in Manam and Nurai.

27. DISTRIBUTION OF NEI KURI

TABLE-27 DISTRIBUTION OF NEI KURI

Sl. No.	Nei Kuri	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
2.	Pitha vaatha Neer	11	55%	13	65%
3.	Pitha Kapha Neer	9	45%	7	35%
Total		**20**	**100%**	**20**	**100%**

FIGURE-27 DISTRIBUTION OF NEI KURI



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According to Table and Figure no 27,the 20 OP patients showed 55% of Pitha vatha Neer; 45% of Pitha Kapha Neer. Among 20 IP patients showed 65 of Pitha vatha Neer ; 35% of Pitha Kapha Neer was affected in Akkini selathumm.

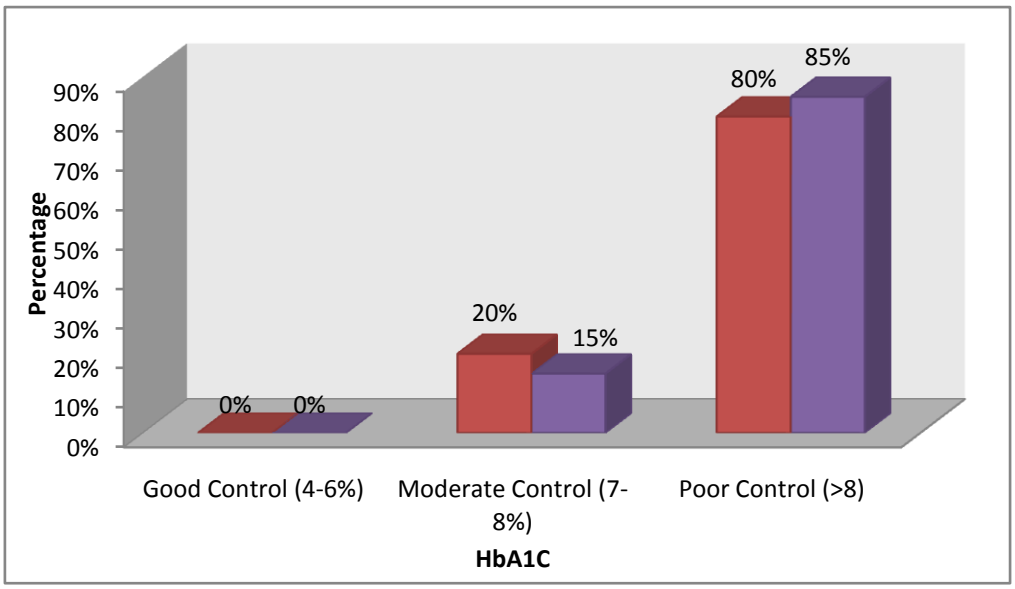
28. DISTRIBUTION OF LABORATORY ANALYSIS

28 (a) HbA1C

TABLE-28(a) PERCENTAGE OF **HbA1C**

Sl. No.	HbA1C	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Good Control (4-6%)	-	-	-	-
2.	Moderate Control (7-8%)	4	20%	8	40%
3.	Poor Control (>8)	16	80%	12	60%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-28(a) PERCENTAGE OF HbA1C



The table 28(a) is showed among 20 out patients 80% had poor control and 20% had fair control, among 20 In patients 60% had poor control and 40% had moderate control of HbA1C in recruitment of study.

28 (b) LIPID PROFILE

TABLE-28(b) LIPID PROFILE

Sl. No.	Lipid Profile	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Total Cholesterol				
	Normal (>200)	-	-	6	30%
	Borderline high (200-239)	10	50%	9	45%
	High (>240)	10	50%	5	25%
2.	LDL Cholesterol (mg/dl)				
	Normal (<100)	1	5%	1	5%
	Above optimal (100-129)	-	-	9	45%
	Borderline High (130-159)	12	60%	8	40%
	High (160-189)	7	35%	1	5%
	Very High (>190)	-	-	1	5%
3.	HDL Cholesterol (mg/dl)				
	Low (<40)	10	50%	8	40%
	Normal (40-60)	10	50%	10	50%
	High (>60)	-	-	2	10%
4.	Triglyceride (mg/dl)				
	Optimal (<150)	2	10%	6	30%
	Borderline (150-199)	10	50%	7	35%
	High (200-499)	8	40%	7	35%
	Very High (>500)	-	-	-	-
5.	VLDLCholesterol (mg/dl)				
	Normal (<40)	11	55%	15	75%
	High (>40)	9	45%	5	25%

A major ratio of the 40 patients included in the study had total cholesterol within the range of borderline and LDL level was above optimal,triglycerides is borderline and HDL and VLDL are normal.

29. CHANGES IN DIAGNOSTIC FINDINGS BEFORE AND AFTER TREATMENT

29(a) Changes in Electro diagnostic findings and Neuro pathic - pain score levels

Table 29(a): Electro diagnostic findings of patients with Diabetic neuropathy before and after treatment (N = 8)

Electro diagnostic study	Mean ± SD (Before)	Mean ± SD (After)	P value
NCV- Peroneal Nerve (m/sec)	44.87 ± 4.38	46.37 ± 4.35	0.041
NCV- Tibial Nerve (m/sec)	41.75±3.37	45.35±5.63	0.018
Distal Latency- Sural Nerve (millisecond)	3.25 ±0.39	3.00 ±0.43	0.017
Sensory Amplitude- Sural Nerve (microvolt)	12.12± 6.53	13.00± 5.99	0.109
Motor Amplitude- Proneal Nerve (microvolt)	1.55± 1.08	1.91 ±1.16	0.018
Motor Amplitude- Tibial Nerve (microvolt)	2.17 ±1.03	2.78 ±0.08	0.016

Table 29 (a) was showed Significant improvements were documented in nerve conduction velocity and motor amplitude of common peroneal and tibial nerves. In addition, the distal latency of sural nerve was significantly improved after three months.

29(b)- NEUROPATHIC PAIN SCORE

29.b DISTRIBUTION OF NEUROPATHIC-PAIN SCORE

TABLE 29 (c) NEUROPATHIC-PAIN

TABLE 29(d) NEUROPATHIC-PAIN

SCORE CHART FOR IN-PATIENT

O/P NO	AGE	NEUROPATHIC-PAIN SCORE				I/P NO	AGE	NEUROPATHIC-PAIN SCORE		
		DURING 1ST VISIT	FOLLOWING 1 month	END OF THE TREATMENT				DURING 1ST VISIT	FOLLOWING 1 month	END OF THE TREATMENT
38680	55	8	2	3		1281	55	7	1	2
38599	59	7	1	2		1324	59	7	2	3
38871	60	8	4	3		1379	60	8	2	4
39099	59	7	3	2		1391	59	7	1	2
41759	58	8	3	5		1652	58	8	2	2
42633	61	6	3	4		1804	61	8	1	2
44668	52	7	1	2		1926	52	7	1	3
44644	40	5	1	2		1942	40	6	2	1
45076	54	8	2	3		2036	54	6	2	3
49053	49	8	2	3		2162	49	5	2	2
50529	65	9	4	7		2300	65	5	1	1
51787	58	7	1	2		2397	58	7	1	2
52435	50	9	2	3		2711	50	8	2	3
68789	40	6	1	2		2783	40	6	1	2
63112	42	7	1	2		2810	42	5	0	1
63732	54	7	1	2		1379	54	7	1	2
68002	65	7	2	3		3014	65	5	1	2
103590	56	6	1	2		351	56	5	1	2
11871	59	7	2	2		458	59	7	2	3
14898	52	5	1	2		459	52	8	2	3

SCORE CHART FOR OUT-PATIENT

FIGURE 29(c) NEUROPATHIC-PAIN SCORE OUT-PATIENT

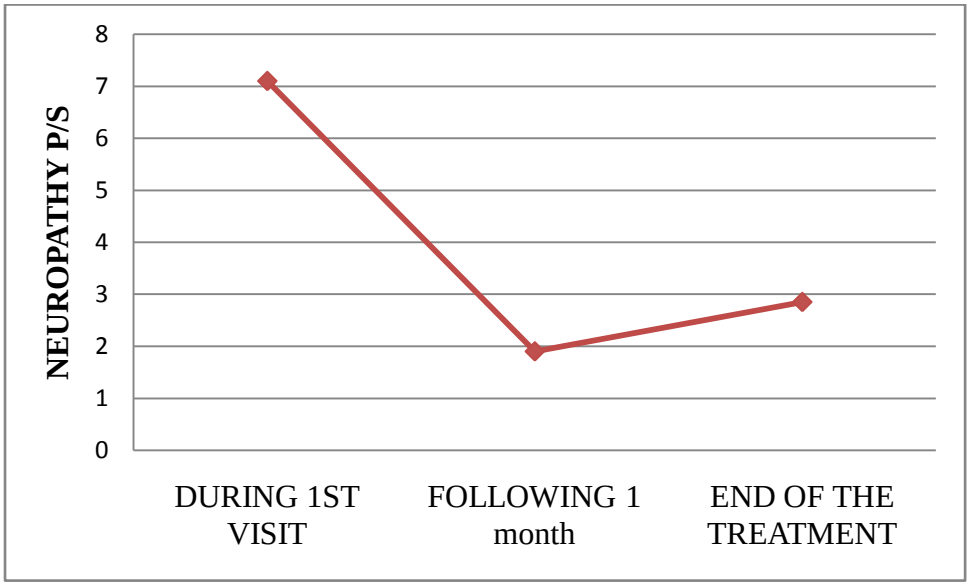


FIGURE 29(d) NEUROPATHIC-PAIN SCORE IN-PATIENT

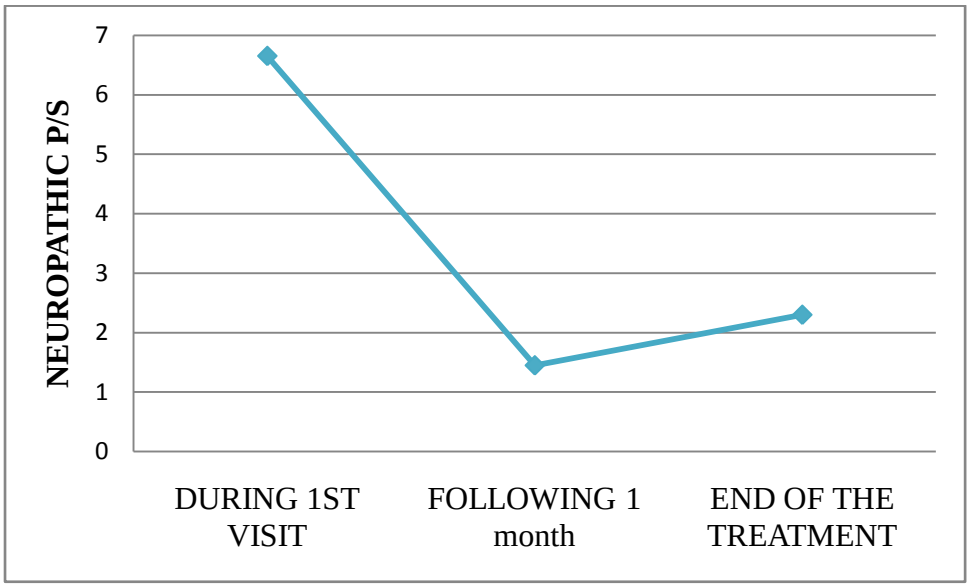


Table and FIG 29(c,d) was showed, Detailed information on patients' basic characteristics is summarized Neuropathic pain scores reduced significantly one month and three months after the intervention.

OUT-PATIENT CASE SHEET: 20 PATIENTS TREATED IN OP FOR AKKINI SELATHUMAM

Sl. No.	OP No.	Name	Age/ Sex	Occupation	Duration of Illness	Date of Onset of Treatment	Date of Tremination of Treatment	Total Days	Results
1.	38680	V.VEALLAMBAL	55/F	House wife	5 Years	30.04.2018	29.07.2018	90	Good
2.	38599	S.PERUMAL	59/M	Business	6 Years	30.4.2018	2.08.2018	92	Good
3.	38871	S.MUMTAJ	60/F	House wife	1 Year	01.5.2018	05.08.2018	93	Good
4.	39099	P.SUBRAMANI	59/M	Business	3 Years	2.5.2018	01.08.2018	90	Moderate
5.	41759	K.RAJASUNDARY	58/F	Police	3 Years	11.5.2018	08.08.2018	91	Good
6.	42633	V.SORNAM	61/F	House wife	1 Year	14.5.2018	21.08.2018	97	Good
7.	44668	K.SELLAMMAL	52/F	Post master	5 Years	22.5.2018	26.08.2018	96	Good
8.	44644	T.NALLASAMY	40/M	Police	6 Years	22.5.2018	20.08.2018	91	Good
9.	45076	S.JAYA	54/F	House wife	2.5 Years	23.5.2018	22.08.2018	91	Good
10.	49053	G.KASIVISVANATHAN	49/M	Clark	3 Years	08.06.2018	08.09.2018	90	Moderate
11.	50529	T.THANKAMANI	65/F	Beedi maker	5 Year	14.06.2018	13.09.2018	90	Good
12.	51787	V.SAROJA	58/F	House wife	3 Years	19.07.2018	16.10.2018	88	Good
13.	52435	K.POOLAMBAL	50/F	Beedi maker	3 Years	21.6.2018	26.09.2018	97	Moderate
14.	68789	U.SATHEESKUMAR	40/M	Driver	1 Year	28.07.2018	02.11.2018	95	Good
15.	63112	T.BEARNAD	42/M	Welding worker	1 Year	30.07.2018	4.11.2018	95	Good
16.	63732	T.SHANKAR	54/M	Clark	2 Years	1.08.2018	06.11.2018	97	Good
17.	68002	S.KOMATHY	65/F	House wife	1 Year	16.08.2018	19.11.2018	94	Good
18.	103590	K.SIVASAMY	56/M	Business	2 Years	14.12.2018	15.03.2018	92	Good
19.	11871	P.RAMACHANDRAN	59/M	Teacher	4 Year	1.02.2019	02.05.2018	92	Good
20.	14898	T.RAMALUXMY	52/F	Housewife	3 Years	9.02.2019	11.05.2018	92	Good

IN-PATIENT CASE SHEET: 20 PATIENTS TREATED IN IP FOR Akkini selathumam

Sl. No.	IP No.	Name	Age/Sex	Occupation	Duration of Illness	Date of Admission	Date of Discharge	No.of Days		Total Days	Results
								IP	OP		
1.	1281	T.MARUTHAVALLI	68/F	Labour	6 years	12.5.2018	21.6.2018	39	51	90	Moderate
2.	1324	V.SANKARAPANDY	66/M	Business	2 years	18.5.2018	03.6.2018	15	75	90	Good
3.	1379	K.THEVATHASH	68/M	Painter	5 years	24.5.2018	28.6.2018	35	60	85	Good
4.	1391	T.JANAKI	54/F	Housewife	3 years	26.5.2018	25.6.2018	30	57	87	Moderate
5.	1652	V.AARUMUGAM	68/M	Clerk	3 years	26.6.2018	25.7.2018	31	52	83	Good
6.	1804	P.RANJITHM	60/F	Housewife	6 years	16.7.2018	15.8.2018	30	62	92	Good
7.	1926	T,JESUMANI	60/F	farmer	2years	28.7.2018	14.9.2018	16	66	82	Moderate
8.	1942	K.SELLATHURI	49/M	cooley	3 years	30.7.2018	10.9.2018	41	49	90	Moderate
9.	2036	K.NALLAMBAL	48/F	Housewife	2 years	07.8.2018	11.9.2018	34	51	85	Good
10.	2162	R.ESAKKIAMBL	63/F	Housewife	6 years	23.8.2018	8.10,2018	46	44	90	Poor
11.	2300	T.SELVI	40/F	Agri.Labour	2 years	07.9.2018	09.10.2018	32	53	85	Good
12.	2397	K.POOMANI	65/F	Housewife	5 years	22.9.2018	15.10.2018	22	58	80	Moderate
13.	2711	S.MANONMANI	60/F	Cook	4 years	8.11.2018	27.11.2018	20	65	85	Moderate
14.	2783	T.PAARVATY	60/F	Housewife	3 years	15.11.2018	7.12,2018	22	68	83	Good
15.	2810	T,AVUDITHI	58/F	Cooley	5 years	17.11.2018	16.12,2018	29	59	88	Good
16.	1379	S.THEVATHS	68/M	farmer	6 years	23.11.2018	12.12.2018	20	60	80	Good
17.	3014	A.RAJIYA	50/F	Cooley	4 years	10.12.2018	01.1.2019	22	60	82	Moderate
18.	351	V,RAJAMMAL	59/F	Housewife	5 years	13.2.2019	13.03.2019	31	57	88	Moderate
19.	458	T.SUBRAMANIJAM	60/M	Ret.Police	5years	23.2.2019	23.3.2019	30	62	92	Good
20.	459	S,NANKAYAR	55/F	Housewife	3 years	23.2.2019	23.3.2019	30	60	90	Moderate

INVESTIGATION CHART FOR OUT-PATIENT: BLOOD INVESTIGATIONS

Sl. No.	OP No.	BEFORE TREATMENT					AFTER TREATMENT				
		Hb	TC	DC			Hb	TC	DC		
				P	L	E			P	L	E
01.	38680	12.3	8100	60	38	02	12.3	8400	62	28	10
02.	38599	11.6	7000	65	32	03	10.4	6400	60	36	04
03.	38871	10.3	7500	52	45	03	12.3	7000	68	28	04
04.	39099	10.6	9000	70	26	04	11.2	8800	70	28	02
05.	41759	10	7600	62	36	02	12.1	7500	60	36	04
06.	42633	14.4	9000	62	32	02	10.8	8000	62	33	05
07.	44668	11.7	5500	62	35	03	11.9	6200	63	34	03
08.	44644	9.4	8400	64	32	04	11.6	4.2	64	33	03
09.	45076	12.5	7000	62	34	04	13	7000	62	34	04
10.	49053	10.5	6400	60	36	04	13	7300	61	26	03
11.	50529	10.2	7500	64	29	04	10	7400	62	36	02
12.	51787	12.8	8000	65	29	06	12	8000	64	30	06
13.	52435	12.9	6400	64	32	04	13	7200	65	32	03
14.	68789	10.9	8800	56	40	04	11	8300	58	40	02
15.	63112	12.6	7800	65	31	04	11.6	7500	66	30	04
16.	63732	9.8	9000	59	37	04	10.2	8700	60	38	02
17.	68002	13	8400	68	30	02	12.5	8200	68	30	02
18.	103590	10.3	7500	52	45	03	12.3	7000	68	28	04
19.	11871	11.4	8100	60	36	04	11.8	7300	64	30	06
20.	14898	13.5	8500	64	31	02	13	8500	64	31	03

Hb-Hameoglobin,TC- Total White Cell Count, DC- Differential Count White Cell Count, P-Polymorph, L-Lymphocyte, E-Eusinophil

INVESTIGATION CHART FOR IN-PATIENT: BLOOD INVESTIGATIONS

Sl. No.	IP No.	BEFORE TREATMENT					AFTER TREATMENT				
		Hb	TC	DC			Hb	TC	DC		
				P	L	E			P	L	E
1.	1281	11.6	7000	65	32	03	10.4	6400	60	36	04
2.	1324	12.5	8000	64	30	03	13	8500	64	31	03
3.	1379	11	9100	54	44	02	12.0	9500	60	33	07
4.	1391	12	8100	64	32	04	12	8000	66	28	06
5.	1652	12.3	8100	60	38	02	12.3	8400	62	28	10
6.	1804	11.5	8100	60	35	05	12.2	8100	60	38	02
7.	1926	12.8	6500	61	35	04	13.8	6500	60	36	04
8.	1942	10.6	7500	62	36	02	11.5	7500	64	32	04
9.	2036	13.9	9600	55.3	31.6	03	11.7	7000	62	38	00
10.	2162	10.8	8000	66	28	06	11.1	6500	60	34	06
11.	2300	12.6	7800	65	31	04	11.6	7500	66	30	04
12.	2397	12.1	9800	70	26	04	12.0	8900	68	28	04
13.	2711	15	9000	58	38	04	13.2	7200	65	33	02
14.	2783	12	8000	62	36	02	12.5	8000	62	36	02
15.	2810	12	7100	58	37	05	12	7000	60	34	06
16.	1379	10.9	8200	60	35	05	10	8000	62	36	02
17.	3014	12.3	7100	62	32	06	13	8000	58	38	04
18.	351	12	8600	60	24	16	12.4	8100	60	36	04
19.	458	10.5	8800	64	34	06	9.9	7800	64	32	04
20.	459	10.3	6800	58	38	04	10.6	7000	60	38	02

Hb-Hameoglobin,TC- Total White Cell Count, DC- Differential Count White Cell Count, P- Polymorph, L-Lymphocyte, E-Eusinophil

INVESTIGATION CHART FOR OUT-PATIENT: PATIENTS BLOOD SUGAR & URINE SUGAR

Sl. No.	OPNo.	BLOOD TEST						URINE TEST					
		BEFORE TREATMENT			AFTER TREATMENT			BEFORE TREATMENT			AFTER TREATMENT		
		FBS	PPBS	S.U	FBS	PPBS	S.U	ALB	SUGAR	DEPOSITS	ALB	SUGAR	DEPOSITS
1.	38680	170	307	33	140	210	30	Nil	+++	Few Pus cells ++	Nil	+	Nil
2.	38599	213	284	35	106	144	34	Nil	++	Nil	Nil	Nil	Nil
3.	38871	136	191	33.2	125	152	19	Nil	++	Pus cells 10-12	Nil	Nil	Few pus cells +
4.	39099	210	280	20	136	170	20	Nil	+	Nil	Nil	Nil	Nil
5.	41759	276	378	25	202	253	23	Nil	++	Nil	Nil	+	Nil
6.	42633	194	227	33	180	196	13	Nil	++	Few pus cells ++	Nil	Nil	Nil
7.	44668	172	272	28	155	204	30	Nil	+	Nil	Nil	Nil	Nil
8.	44644	138	202	30	125	175	25	Nil	++	Pus cells 10-12	Nil	Nil	Nil
9.	45076	178	245	30	150	198	30	Nil	+	Nil	Nil	Nil	Nil
10.	49053	203	267	20	144	200	18	Nil	+	Few pus cells ++	Nil	Nil	Nil
11	50529	208	298	23	175	204	21	Nil	++	Nil	Nil	Nil	Nil
12.	51787	191	270	30	180	204	26	Nil	++	Nil	Nil	Nil	Nil
13.	52435	291	315	18	233	218	29	Nil	+++	Nil	Nil	++	Nil
14.	68789	199	242	29	152	198	27	Nil	++	Nil	Nil	Nil	Nil
15.	63112	150	288	30	132	204	28	Nil	Nil	Nil	Nil	Nil	Nil
16.	63732	170	330	24	162	220	30	Nil	++	Nil	Nil	+	Nil
17.	68002	169	304	22	151	209	26	Nil	+	Nil	Nil	Nil	Nil
18.	103590	201	304	30	135	213	26	Nil	+	Nil	Nil	Nil	Nil
19.	11871	178	244	26	132	194	27	Nil	Nil	Nil	Nil	Nil	Nil
20.	14898	139	191	23	129	144	26	Nil	Nil	Nil	Nil	Nil	Nil

FBS-Fasting Blood Sugar, PPBS-Post Parandial Blood Sugar, S.U-Serum Urea, ALB-Albumi

INVESTIGATION CHART FOR IN-PATIENT: PATIENTS BLOOD SUGAR & URINE SUGAR

Sl. No.	IP No.	BLOOD TEST						URINE TEST					
		BEFORE TREATMENT			AFTER TREATMENT			BEFORE TREATMENT			AFTER TREATMENT		
		FBS	PPBS	S.U	FBS	PPBS	S.U	ALB	SUGAR	DEPOSITS	ALB	SUGAR	DEPOSITS
1.	1281	205	205	26	95	105	26	Nil	++	Nil	Nil	Nil	Nil
2.	1324	204	204	24	122	268	28	Nil	++	Pus cells ++	Nil	Nil	Nil
3.	1379	192	192	22	135	204	25	Nil	Nil	Nil	Nil	Nil	Nil
4.	1391	213	213	32	145	237	34	Nil	+++	Nil	Nil	+++	Nil
5.	1652	160	160	26	132	202	28	Nil	+	Nil	Nil	+	Nil
6.	1804	261	261	19	264	288	26	Nil	+	Nil	Nil	Nil	Nil
7.	1926	186	186	26	113	260	28	Nil	+	Pus cells ++	Nil	+	Pus cells ++
8.	1942	179	379	12	160	260	25	Nil	+	Nil	Nil	Nil	Nil
9.	2036	182	182	22	160	217	21	Nil	++	Pus cells ++	Nil	+	Pus cells ++
10.	2162	185	185	34	138	204	27	Nil	+++	Pus cells ++	Nil	++	Nil
11.	2300	174	274	26	155	270	24	Nil	+	Nil	Nil	+	Nil
12.	2397	135	135	30	130	223	26	Nil	+++	Pus cells ++	Nil	++	Nil
13.	2711	237	237	35	220	300	38	Nil	+++	Nil	Nil	Nil	Nil
14.	2783	192	192	26	188	240	28	Nil	Nil	Nil	Nil	Nil	Nil
15.	2810	119	119	28	110	190	26	+	+++	5-7 pus cells	Nil	+	Nil
16.	1379	198	198	26	175	235	30	Nil	+	Pus cells ++	Nil	Nil	Nil
17.	3014	174	174	28	143	204	26	Nil	Nil	Nil	Nil	Nil	Nil
18.	351	135	135	30	95	127	28	Nil	+	Nil	Nil	Nil	Nil
19.	458	240	240	24	202	280	46	Nil	+	Nil	Nil	Nil	Nil
20.	459	220	220	28	190	310	30	Nil	+	Pus cells ++	Nil	Nil	Nil

FBS-Fasting Blood Sugar, PPBS-Post Parandial Blood Sugar, S.U-Serum Urea, ALB-Albumin

INVESTIGATION CHART FOR OUT-PATIENT: PATIENTS HbA1C & LIPID PROFILE

Sl.No	OPNo	BEFORE TREATMENT						AFTER TREATMENT					
		HbA1C	LIPID PROFILE					HbA1C	LIPID PROFILE				
			TC	HDL	LDL	VLDL	TGL		TC	HDL	LDL	VLDL	TGL
1.	38680	9.2	242	40.1	149.1	52.8	264.1	8.7	180	48	99	33	165
2.	38599	8.7	226	39	136	52	260	8.5	210	68	120	22	110
3.	38871	8.7	270	54	183	83	416	8.3	219	54	140	25	127
4.	39099	10	263	42	131	48	242	9.7	202	33	125	56	280
5.	41759	10	238	26	159	53	263	9.7	176	52	110	32	158
6.	42633	8.7	253.2	39.2	174.4	39.6	198.1	8.5	197	67	109	21	105
7.	44668	9.4	267	36	184	47	233	9.2	130	42	82	45	133
8.	44644	8.9	208	40	154	14	170	8.5	211	65	121	25	127
9.	45076	9.4	223	42	131	12	149	9.2	190	45	126	12	110
10.	49053	9.7	247	42	160	16	182	9.5	203	66	120	17	84
11.	50529	10	219	48	141	30	169	9	185	40	180	15	75
12.	51787	9.4	240	30	169	20	195	9.3	213	42	128	12	153
13.	52435	9.9	213	30	68	95	475	9.8	223	57	91	80	398
14.	68789	8.1	247	60	158	29	193	8	167	43	82	32	121
15.	63112	8.9	284	57.2	193	34	168	8.2	190	58	102	33	175
16.	63732	9.2	254	35	195	24	171	9	154	35	95	24	121
17.	68002	9.3	223	44.6	132	47	235	9.1	145	38	86	21.8	109
18.	103590	7.6	224	44	158	22	110	7.3	210	61	89	28	98
19.	11871	8.3	220.5	34.2	158.4	27.9	139.9	8.1	158	61	79	18	88
20.	14898	9.8	211	44	130.3	36.1	144	9.23	166	39	95	46	76

TC-Total Cholesterol, HDL-High Density Lipoprotein, LDL-Low Density Lipoprotein, TGL-Triglyceride

INVESTIGATION CHART FOR IN-PATIENT: LIPID PROFILE AND HbA1C

Sl. No.	IP No.	BEFORE TREATMENT						AFTER TREATMENT					
		HbA1C	LIPID PROFILE					HbA1C	LIPID PROFILE				
			TC	HDL	LDL	VLDL	TGL		TC	HDL	LDL	VLDL	TGL
1.	1281	7.7	196	39	127	23	118	6.8	192	50	122	20	102
2.	1324	9.8	278	38	119	21	105	9.6	192	46	102	28	120
3.	1379	9.1	190	39	117	23.6	392	8.7	192	50	122	20	102
4.	1391	8.1	226	30	117	78.4	392	7.9	154	40	84	30	115
5.	1652	8.8	227	39	136	52	260	8.1	215	46	146	23	75
6.	1804	8.5	175	48	103	24	170	8.3	181	52	96	26	120
7.	1926	8.5	275.3	35	122.8	17.4	87.1	8.3	163	72	66	25	127
9.	1942	8.0	190	45	118	58	110	7.09	181	50	150	31	186
10.	2036	9.1	227	61	142.4	23.6	118	7.9	200	44	128	27	157
11.	2162	7.3	181	50	150	31	186	7.1	154	40	84	30	150
12.	2300	9.23	206	31	190	49	190	8.2	166	39	111	55	76.3
13.	2397	7.1	230	54	152	24	178	6.8	230	54	132	34	118
14.	2711	7.7	240	58	236	46	232	7.5	223	36	140	47	236
15.	2783	7.3	191	37.4	129.94	23.66	118.3	7.3	180	30	137	13	67
16.	2810	8.7	205	57	119	29	145	8.3	201	54	97	17	98
17.	1379	9.3	230	54	152	24	178	9.2	168	46	88	34	171
18.	3014	8.3	270	50	150	20	180	8.2	208	48	106	17	186
8.	351	7.8	182	48	98	30	176	7.7	175	58	93	25	113
19.	458	9.8	255	72	150	33	151	9.7	240	80	130	30	148
20.	459	9.1	201	53	126	22	112	8.9	210	58	102	25	138

TC-Total Cholesterol, HDL-High Density Lipoprotein, LDL-Low Density Li poprotein, TGL-Triglyceride

BMI CHART FOR OUT-PATIENTS (OP)

Sl. No.	OP No.	Name	Age/ Sex	BMI					
				Before Treatment			After Treatment		
				WT	HT	BMI	WT	HT	BMI
1.	38680	V.VEALLAMBAL	55	63	160	24.61	62	160	24.2
2.	38599	S.PERUMAL	59	110	167	39.44	105	167	37.6
3.	38871	S.MUMTAJ	60	65	149	29.28	65	149	29.3
4.	39099	P.SUBRAMANI	59	80	165	29.38	76	165	27.9
5.	41759	K.RAJASUNDARY	58	45	142	22.32	46	142	22.8
6.	42633	V.SORNAM	61	93	170	32.18	91	170	31.5
7.	44668	K.SELLAMMAL	52	63	152	27.27	62	152	26.8
8.	44644	T.NALLASAMY	40	75	173	25.06	74	173	24.7
9.	45076	S.JAYA	54	55	145	26.16	55	145	25.9
10.	49053	G.KASIVISVANATHAN	49	65	161	25.08	63	161	24.3
11.	50529	T.THANKAMANI	65	60	160	23.44	59	160	23
12.	51787	V.SAROJA	58	66	143	32.28	65	143	31.8
13.	52435	K.POOLAMBAL	50	56	154	23.61	55	154	23.2
14.	68789	U.SATHEESKUMAR	40	76	179	23.72	73	179	22.8
15.	63112	T.BEARNAD	42	72	174	23.78	70	174	23.1
16.	63732	T.SHANKAR	54	65	168	23.03	65	168	23
17.	68002	S.KOMATHY	65	65	149	29.28	63	149	28.4
18.	103590	K.SIVASAMY	56	91	173	30.41	90	173	30.1
19.	11871	P.RAMACHANDRAN	59	60	164	22.31	59	164	21.9
20.	14898	T.RAMALUXMY	52	64	154	26.99	63	154	26.6

WT-Weight, HT-Height, BMI-Body Mass Index, WC-Waist circumference, HC-Hip circumference, WHR-Waist Hip Ratio

BMI CHART FOR IN-PATIENTS (IP)

Sl.No.	OPNo.	Name	Age/ Sex	BMI					
				Before Treatment			After Treatment		
				WT	HT	BMI	WT	HT	21.9
01.	1281	T.MARUTHAVALLI	68	54	154	22.77	52	154	28.4
02.	1324	V.SANKARAPANDY	66	88	172	29.75	84	172	27
03.	1379	K.THEVATHASH	68	82	172	27.72	80	172	26.6
04.	1391	T.JANAKI	54	78	170	26.99	77	170	22.4
05.	1652	V.AARUMUGAM	68	58	161	22.38	58	161	26.2
06.	1804	P.RANJITHAM	60	60	150	26.67	59	150	21.2
07.	1926	T,JESUMANI	60	45	149	20.27	47	149	17.2
08.	1942	K.SELLATHURI	49	50	169	17.51	49	169	27.5
09.	2036	K.NALLAMBAL	48	75	163	28.23	73	163	32.4
10.	2162	R.ESAKKIAMBAL	63	60	136	32.44	60	136	23.7
11.	2300	T.SELVI	40	70	168	24.8	67	168	22.3
12.	2397	K.POOMANI	65	56	160	21.88	57	160	27.3
13.	2711	S.MANONMANI	60	60	147	27.77	59	147	25.8
14.	2783	T.PAARVATHY	60	55	146	25.8	55	146	28
15.	2810	T,AVUDITHAI	58	65	150	28.89	63	150	30.8
16.	1379	S.THEVATHAS	68	75	155	31.22	74	155	30.8
17.	3014	A.RAJIYA	50	57	150	25.33	56	150	24.9
18.	351	V,RAJAMMAL	59	43	157	17.44	45	157	18.3
19.	458	T.SUBRAMANIJAM	60	63	168	22.32	62	168	22
20.	459	S.NANKAYAR	55	53	163	19.95	52	163	19.6

CHAPTER-V

DISCUSSION

The Akkini selathumam is correlated in modern science is Diabetic Neuropathy, which affects up to 50% of type 2 diabetic patients. Whereas some patients may have extremely painful symptoms, others with a more marked neuropathic deficit may be asymptomatic. Diagnosis requires careful examination of the lower limbs. Uncontrolled long standing diabetes is the main cause for developing the diabetic complications. This clinical study is designed to target the risks of macro-vascular complications ensuing diabetes and to improve the quality of living of the diabetic individuals, with an efficacious herbal formulation mentioned in Siddha literature.

In this study, 40 type-II diabetic patients who are attending the OP and IP at the Dept. of Pothu Maruthuvam, Govt. Siddha Medical College and Hospital, Palayamkottai were randomly selected for carried out this study. 20 screened and selected patients were admitted as Inpatients in Post Graduate Department of Pothu Maruthuvam and were treated with the trial medicine. After discharge all the twenty patients were followed as the Outpatients. 20 Out patients in the Outpatients Department of Pothu Maruthuvam were also treated with the trial drug. From my entire study the following findings are observed.Details are given below,

1. Distribution of Gender

The subjects included females 65% in OP, 50% in IP and males 35% in OP and 50% in IP were affected proving gender disparity in the onset of the disease. The female population was more than the male counterpart. This compares well with a study on WHO global data which stated that the prevalence ratio of diabetes between men and women varies markedly, with no consistent trend. The ADA stated the relative difference in frequency between the sexes is probably related to the presence of underlying factors, such as post-delivery leads to obesity, rather than to a sex-specific genetic tendency.

2. Distribution of Age

The highest incidence of Akkini selathumam is among the age group of 51-60 years, closely followed by 61-70 years. From this study, it was observed that the

diabetic subjects were averagely older. This shows type-II diabetes begins typically in middle life or later, the prevalence rises with age.

3. Distribution of Educational Status

The assessment of patient's knowledge showed the majority of patients were middle(35%) and high school(45%) educated.The educational level had no impact on glycemic control, but the patients of high educational level had better awareness of the complications and a high rate of adherence to diet.

4. Distribution of Occupation

The highest (55%) incidence of Akkini selathuma is among OP and IP patients were housewives, it belongs to female counterpart.

5. Distribution of Religion

The majority of patients were Hindus among OP and IP as 80%. The highest incidence is among the Hindu population, which is the major population in India. Diet and lifestyle of rituals may influence the control of blood sugar and dyslipidaemia.

6. Distribution of Marital Status

The highest incidence among the OP and IP patients were married. Pregnancy and obesity can influence the married status in Akkini selathumam.

7. Distribution of Clinical Manifestation

Diabetes is a chronic illness that requires continuing medical care and patient self-management education to prevent acute complications and to reduce the risk of long-term complications.The data from the observation showed 100% of incidence of polyuria, polydipsia, Polyphagia and nocturia in both In Patients and out-patients.

Diabetic neuropathy itself usually causes Numbness,Burning sensation of the limb, Tingling Sensation and pain . These features also(100%) present among OP & IP patients in who were recruited for this study.

8. Distribution of Mode of Onset

The onset of Akkin Selathumam ensures a chronic mode of onset with relative percentage of 75% in OP and 95% in IP. The percentage of recently found supports the fact that in many diabetics the disease is first detected when the patient presents with a complications.

9. Distribution of Duration of Illness

Akkini Selathumam is greatly noticed in 40 patients were suffering more than 3 to 6 years are more suffering with Madhumegam.

It was observed that duration of type-II diabetes had a positive correlation with glycated haemoglobin. This is because the body becomes more resistant to insulin with increasing duration of diabetes.

10. Distribution of Family History

Among the Outpatients, 65% of the patients had positive family history and 35% of the patients had negative family history in In patients 60% had negative family history. Family history is high prevalence to got diabetic complications.

11. Distribution of Previous Treatment

Among OP and IP patients of 90% and 95% were taken previous therapy. Majority of patients despite of previous therapy for madhumegam had developed Akkini Selathumam, reflecting the resistance developed to the undertaken therapy.

12. Distribution of Personal History

The observations illustrate that the disease was majority of patients were taken mixed diet. According Yugi Vaidhya Chinthamani, the dietary factors that cause the disease are taking excessive consumption of non-vegetarian diet. Here the observations coincide with Yugi's concept.

Although regulation of blood glucose and lipid profile to achieve near normal levels is a primary goal in the management of diabetic dyslipidaemia, and thus, dietary techniques that limit hyperglycaemia following a meal are important in limiting the complications of diabetes.Indian diets rich in carbohydrate and low in Omega -3PUFA exacerbate hypertriglyceridemia (Srinivas et al.2014).

Distribution of Socio economic Status

Among the Out-patients 45% belonged to the lower middle Socio-Economic status and 35% belonged tolower middle and poor group.

This Observation indicates no class variation in the manifestation of the disease. But the low income should influence the awareness of diet, health care and routine medical check-up of patients.

13. Distribution of Other System Involvement

Both In patients, Out Patients had Respiratory system, cardiovascular system and musculo skeletal system(90%) affected. Among this majority of cases affected with musculo skeletal system in OP and IP patients. The course may be due to degenerative changes.

14. Body Mass Index

According to WHO 60% of the quality of an individual's health depends on his/her diet and regimens. Early adoptions of healthy habits can problems of future years. Present study reflected that there was a higher prevalence of overweight and obesity among OP and IP patients (65% & 85%). Increased BMI is a risk factor of diabetic related complications.

15. Distribution of Constitution of Body

Among, 40 Outpatients and Inpatients 100% were affected in thontha thegi. The study reflected that higher prevalence was Pittham with other doshas.

16. Distribution of Gunam

100% of all 40 patients had Rajogunam

17. Distribution of Kaalam

The maximum numbers of cases were treated in their pitha kaalaM(80%) among OP and IP patients. Siddha text also denoted the pittha kaalam is more prevalence to Akkini selathumam.

Paruva Kaalam

There was high incidence of this study reflected munpanikaalam (75%) among OP patients and pinpanikaalam (85%) among IP patients. The text book of Siddha maruthuvam stated elavenil and mudhuvenil kaalam are more prevalence for Madhumegam occurs.

Akkini selathumam is the complications or sequence of Madhumega disease. Therefore it could not counterpart of the disease origin.

18. Thinai

Among, Outpatients 90% belonged to Marutham (i.e. Plain & its surroundings) and 85% in Inpatients.

19. Mukkutram

a. Derangement of Vatham

Abanan, Viyanan, Samanan and Uthanan were mostly affected in all the 100% of the Outpatients and Inpatients. Kirukaran was affected in 75% of the Out and In- patients . Piranan was affected in 5% of the Outpatients and 7% of the Inpatients; Pranan maintains the life force in a normal healthy body, this vayu when affected causes fatigue as presentd along with Akkini selathumam.

- Abanan is the responsible for excretion of urine and motion. This vayu is affected in this disease causing constipation and polyuria.
- Viyaanan functions to induce normal physiological movements in the body. This vayu is affected leading to decreased activity due to tiredness, claudication pain and weakness.
- Uthanan is responsible for speech, strength of the mind and the body. Since there is a decrease in the strength of the body and mind.
- Altered Samanan is leads to polyphagia and indigestion.
- Kirukaran is responsible for appetite which is affected in this disease.

b.Derangement of Pitham

Analagam, Ranjagam and Sathagam were affected in 100% of both Inpatients and Outpatients. Prasagam was affected in 30% of the Outpatients and 35% of the Inpatients. Alosagam was affected in 25% of the Outpatients and 5% of the Inpatients.

- Analagam is responsible for appetite. Since there is excess appetite or loss of appetite among the patients.
- Ranjagam contributes to the normal function of the blood components. This pitham is affected since there is a decreased haemoglobin level and increased burning sensation in Akkini selathumam.
- Sathagam enables the performance of the intended actions if altered causing tiredness.
- Prasagam gives lustre to the skin, which is affected also.
- Altered alosagam causing blurring of vision.

c. Derangement of Kapham

In types of Kapha dosham, Avalambagam and Kilethagam were affected in all the 100% of both Inpatients and Outpatients. Santhigam was affected in 65% of Outpatients and 75% of Inpatients.

- Avalambagam resides in the lungs and helps the other four types of kapham to function in equilibrium. Since the equilibrium is altered due to involvement of other forms of kapham also affected.
- Deranged Kiethagam excessive appetite or loss of appetite is present.
- Santhigam resides in the joints and helps in its movement. Since there was joint pain,

20. Involvement of Ezhu Udalthathukkal

In ezhu udal Kattugal, Saaram and Senneer was affected in all 100% of the Outpatients and Inpatients. Oon was affected in 65% of the Out patients and 15% of the Inpatients. The kozhuppu was affected in 80% of the Outpatients and 85% of the Inpatients. Enbu was affected in 20% of the out-patients and of the 25% Inpatients and sukkilam was affected in 5% of Outpatients only.

- Saaram strengthens the body and mind, since, there is loss of appetite and strength less causing body tiredness the first thathu is affected.
- Senneer is affected which produces decreased haemoglobin and accelerated atherosclerosis. Oon is responsible for the structural muscular component of the body, this is affected in the weakness caused by stroke. Excessive kozhuppu thathu also causes accelerated atherosclerosis in Akkini selathumam.
- Enbu and Kozhuppu are responsible for the movements of the body and gives lubrication to the joint cavities. There was reflected in masculo skeletal disorders like Oteo arthritis, back pain, shoulder pain etc.,due to obesity, and senility.

21. Kanmenthiriyam

The present study showed kaal and eruvai were affected in majority of cases in OP and IP patients (100% & 40%). It was reflected regarding the altered functions of Kanmenthiriyam due to the sequence of Akkini selathumam.

22. Gnanenthiriyam

Among Outpatients and In patients Mei was affected in 50% and 25% of the cases leads to altered sensation and pain in madhumegam due to altered Viyanan and Devathathan. The pathology also can be overlap with sign and symptoms of diabetic neuropathy causes peripheral vascular diseases, stroke etc.

23. Kosam

Among 40 patients annamaya kosam (100%) was affected due to altered abanan, samana vayu, anaila Pitham and kilethagam, in udalkattukal saaram and senneer.

24. Envagai Thervugal

- Sparisam was affected in 50% of Outpatients and 25% of Inpatients due to altered sensations of pain in claudication and numbness in neuropathy.
- Niram was affected in 30% of Outpatients 35% of Inpatients due to loss of lustre.
- Vizhi was affected in 10% of Outpatients and 20% of Inpatients, blurring of vision due to cataract and aging.
- Malam was affected in 30% of Outpatients and Inpatients due to constipation.

- Naa and Moothiram were affected in Inpatients and Outpatients. There was presence of dryness of tongue due to dehydration and polyuria with excretion of albumin, glucose, and abnormal neerkuri.
- Mozhi was affected 10% among Inpatients only with Akkini selathumam in the study.
- In Naadi examination majority of cases had pitha vatha naadi in OP and IP patients (55% & 65%) pitham kapham were 45% in OP and 35% in IP.

25. Neerkuri

In Neerkuri, Niram was affected in 55% of Out-patients and 45% of Inpatients which the colour was replicated crystal clear urine and indicates asathiyam. Manam was affected like honey odour in 20% of Outpatients and 30% of Inpatients. Nurai was affected in 35% of Outpatients and 25% of Inpatients.

26. Neikuri

In Neikuri, 100% of patients had Thontha neer. Which was reflected some Asathiya Neerkuri such features like conch shape, decoration type, bat shape, bow shape etc.

27. Laboratory Analysis

A. HbA1C

Among the 40 patients recruited for the study most patients in OP & IP (80% & 85%) had poor control (>8%) of HbA1C.

This study reveals high prevalence of hypercholesterolemia, hypertriglyceridemia, high LDL-C and low HDL-C levels among subjects with high HbA1C levels which are well known risk factors for cardiovascular diseases. Insulin affects the liver Apo-lipoprotein production. It regulates the enzymatic activity of Lipoprotein Lipase (LPL) and Cholesterol ester transport protein.

All these factors are likely cause of dyslipidaemia in Diabetes mellitus. Moreover, insulin deficiency reduces the activity of hepatic lipase and several steps in the production of biologically active LPL may be altered in DM.

B. Lipid Profile

Above this study, the pattern of lipid profile parameters in diabetic subjects and it's among the 40 patients included in the study, major ratio of the patients had TC within the range of borderline high (OP-50% & IP-45%) and high (OP-45% & IP-25%), LDL within the range ofborderline high (OP-60% & IP-40%) and high (OP-35% & IP-5%) above optimal were 45% in Inpatients only.

50% of OP and 40% of IP patients had low HDL levels. Both IP & OP patients had 50% normal HDL levels.

High prevalence of TGL in range of borderline (OP-50% & IP-35%) and high (OP-40% & IP-35%) were among 40 patients.

Majority of OP and IP patients had normal levels of VLDL cholesterol.

28. Gradation of results

Good response was found in 70% of Outpatients and 75% of Inpatients. Moderate improvement was found in 20% of Outpatients and in 20% of Inpatients. Poor result was found in 10% of Out patients and 5% of the Inpatients.

Bio statistical analysis showed significant difference in the action of the trial drug in lowering elevated blood sugar levels, HbA1C, total cholesterol, LDL, TGL, and HDL with a 'p' value of $p<0.001$ for blood sugar, $p< 0.001$ for HbA1C, $p<0.001$ for total cholesterol, $p<0.001$ for LDL $p<0.001$, for HDL $p<0.001$ and for TGL $p=0.001$ before and after treatment with the trial drug. The p value for VLDL cholesterol level was not statistically significant, although difference exists in their mean value with SD.

It shows that though statistically significant clinically the trial drug had an effect in reducing the elevated Blood sugar, HbA1C, Total cholesterol, LDL, HDL and triglyceride levels. At the same times good Response was found in Diabetic neuropathic patient.

Anthropometric measurements also showed significant difference in the action of trial drug in lowering body weight and BMI with p value of <0.001.

CHAPTER-VI

SUMMARY

Diabetic neuropathies are neuropathic disorders that are associated with diabetes mellitus. These conditions are thought to result from diabetic microvascular injury involving small blood vessels that supply nerves (vasa nervorum) in addition to macrovascular conditions that can culminate in Akkini selathum (Diabetic neuropathy).

The clinical trial was conducted in selected 40 patients after the including and excluding criteria,who are affected in Akkini selathum, The sex , age and other parameters were screened and used for this study The patients were subjected to investigations based on lab investications in modern aspect as well as Siddha. The trial drug KADUKKAI CHOORANAMwas administered to all selected patients, at a dose of 25mg/kgBW, for a study period of 90 days.

Blood sugar (FBS, PPBS), HbA1c, lipid profile and Neuropathic- pain score was recorded before and after treatment. Siddha diagnostic parameters of Naadi examination and neerkuri, nei kuri were also observed. The blood sugar was observed once in 28 days.

All the cases administered with the trial drug, they were not reported any adverse reactions. Few patients adapted to the medicine and recovered spontaneously, for such patients the textual dosage of the trial drug was followed. In those who had a persistent complaint, the dosage of the trial drug was titrated according to their Body Mass Index.

Significant improvement was observed in almost all the cases included in the study. Symptoms of tiredness, peripheral neuropathy (pain, Burning sensation and numbness),pain due to vascular claudication, gastric disturbances were remarkably reduced. Significant decreases in the mean values of blood sugar levels, HbA1C, Lipid levels BMI and Neuropathic- pain score were noted before and after treatment. For blood sugar and HbA1C the “p” value was $p<0.001$ and “p” value was $p<0.001$ and for total cholesterol “p” value was $p<0.001$, for LDL “p” value was $p<0.001$, for HDL “p” value was $p<0.001$,for TGL “p” value was $p<0.001$for BMI ‘p’ value was

<0.001,Neuropathic-pain score “p” value was p<0.001, which implies that statistically the trial drug has KADUKKAI CHOORANAMis potent anti-hyperglycemic anti-hyperlipidemic and Neuropatic actions.

Biochemical analysis of the trial drug showed the presence of Calcium, starch, ferrous iron, tannic acid, unsaturated compound, reducing sugar and amino acids. The pharmacological analysis showed that the trial drug produced significant changes in, plasma glucose, haemoglobin, glycosylated haemoglobin HbA1C, plasma insulin,Inflamation and lipid profile.

CHAPTER-VII
CONCLUSION

In conclusion, the significant effect of *KADUKKAI CHOORANAM* in diabetic neuropathy.. Trail medicine was showed markedly decreased body weight. In this clinical study 75% of the patients showed good improvement, 17.5% showed moderate improvement and 7.5% showed poor improvement.The moderate and poor response means the patients are not attending in opd and Ipd (or) irregulary taken the medicine.

Clinically analysing the before and after treatment of *KADUKKAI CHOORANAM*, there was a significant decrease in the mean values of different clinical parameters. Neuropathic- pain score was considerably reduced from 6.9 to 2.7. FBS was reduced from 171 to 151 whereas PPBS was reduced from 213 to 172 .Three month average value of HbA1C levels had revealed slight decrease from 8.7 to 8.18 on average.Noticeable changes were also present in average lipid profile values. Average values of HDL were raised from 43 to 50 whereas average LDL levels had shown decrease from 146 to 109 along with down values of TGL from 207 to 142 .BMI ratio had decreased from 25.00 to24.67 which proves the effective management of body weight.

Akkini selathumam is managed by the trial drug *KADUKKAI CHOORANAM*, it is reduced blood sugar, lipid levels and HbA1C along with elevated lipid levels, a biochemical state desirable for the prevention of diabetes and its macrovascular conditions in diabetic neuropathy.

HbA1C can be used as a potential biomarker for predicting Neuropathy in type-II diabetic patients in addition to glycaemic control hence early diagnosis and may be utilized for screening high-risk diabetic patients for timely intervention with Neuropatic pain lowering drugs. To conduct nerve conduction study in few patients, after the study is showed Significant improvements were documented in nerve conduction velocity and motor amplitude of common peroneal and tibial nerves. In addition, the distal latency of sural nerve was significantly improved after three months.

KADUKKAI CHOORANAM is indicates the protective role against diabetic neuropathy related pain. So, kadukkai choornan is to well control diabtes and hyperlipedemia. The secondary effect of *kadukkai choornan* is reducing the diabetic preiperal neuropathic pain.

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ANNEXURE -I

PREPRATION OF THE TRIAL DRUGS

DRUGS;

KADUKKAI

Purification and Preparation of *Kadukkai Chooranam*

Terminalia Chebula matured fruits weare collected and washed with water and then dried under the sunlight. Cut and remove the inner seeds of fully matured dry fruits of Terminalia chebula shade dried and make into powder.

Ingredients of *Kadukkai Chooranam*

TAMIL NAME	BOTANICAL NAME / FAMILY	PART USED	IMPORTANT PHYTOCHEMICALS	ACTIONS	THERAPEUTIC USES IN SIDDHA
Kadukkai	*Terminallia Chebula*	Fruits	Chebulinic acid, Gallic acid, Tannic acid, Ascorbate, Ellagic acid, Streptozotocin-N, Ethanolic acid	Anti diabetic, Anti oxidant, Analgesic, Hypolipidemic activity	Mega disorders, Burning sensation of the upper and Lower limb, Thirst, Liver disease, Cardiac disease, General Debility polyurea.

Dosage	:	**25mg/ Kg/BW/daily two times a day**
Adjuvant	:	**Warm water**
Duration	:	**90 days**
Reference	:	***Gunapadam Mooligai Muthal Pagam Page No.201***
		Kadukkai, Vallaraiyin Thanimanbu Pg.No. 9

GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI

Certificate of Botanical Authenticity

Certified the following plant drug used in Siddha formulation (Internal) **"KADUKKAI CHOORANAM"** for **AKKINI SELATHUMAM** (DIABETIC NEUROPATHY) taken up for Post-Graduation Dissertation Studies by **Dr. RAJENDRAM AJANTHAN**, PG Scholar MD siddha, Department of Pothu Maruthuvam, are correctly identified and authenticated through Visual inspection / Organoleptic Characters / Experience, Education & Training Morphology Microscopically and Taxonomical methods.

Table 1: Ingredients of Kadukkai Chooranam

S.N	Drug	Botanical Name	Family	Parts Used
01	Kadukkai	*Terminallia Chebula*	Combretaceae	Fruits

Station: Palayamkottai
Date 25/01/18.

25/1/18

Authorized Signature

Dr. S. SUTHA, M.Sc.,M.Ed.,Ph.D.,
Associate Professor
Dept. of Medicinal Botany
Govt. Siddha Medical College
Palayamkottai, Tirunelveli - 2.

ANNEXURE-III (A)

BIO-CHEMICAL ANALYSIS OF KADUKKAI

CHOORANAM PREPARATION OF THE EXTRACT

The extract is directly prepared from the trial drug Kadukkai Chooranam.

QUALITATIVE ANALYSIS

Sl. No.	EXPERIMENT	OBSERVATION	INFERENCE
1.	**TEST FOR CALCIUM** 2ml of the above prepared extract is taken in a clean test tube. To this add 2ml of 4% Ammonium oxalate solution.	A white precipitate is formed.	Indicates the presence of **calcium.**
2.	**TEST FOR SULPHATE:** 2ml of the extract is added to 5% Barium chloride solution.	No white precipitate is formed	Absence of Sulphate.
3.	**TEST FOR CHLORIDE** The extract is treated with Silver nitrate solution.	No white precipitate is formed.	Absence of chloride.
4.	**TEST FOR CARBONATE** The substance is treated with concentrated Hcl.	No brisk effervescence is formed.	Absence of Carbonate.
5.	**TEST FOR STARCH** The extract is added with weak Iodine solution.	No blue colour is formed.	Absence of Starch.
6	**TEST FOR IRON FERRIC** The extract is acidified with Glacial acetic acid and Potassium ferro cyanide.	No blue colour is formed.	Absence of ferric Iron
7.	**TEST OF IRON FERROUS** The extract is treated with concentrated Nitric acid and Ammonium thio cynaate solution.	No blood red colour is formed.	Absence of ferrous Iron.
8.	**TEST FOR PHOSPHATE** The extract is treated with Ammonium molybdate and concentrated Nitric acid.	No yellow precipitate is formed.	Absence of phosphate.
9.	**TEST FOR ALBUMIN** The extract is treated with Esbach's reagent.	No yellow precipitate is formed.	Absence of albumin.

Sl. No.	EXPERIMENT	OBSERVATION	INFERENCE
10.	**TEST FOR TANNIC ACID** The extract is treated with Ferric choloride.	Blue black precipitate is formed.	Indicates the presence of **tannic acid.**
11.	**TEST FOR UNSATURATION** Potassium permanganate solution is added to the extract.	It gets decolourised.	Indicates the presence of **unsaturated compound.**
12.	**TEST FOR THE REDUCING SUGAR** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.	Colour change occurs.	Indicates the presence of **reducing sugar.**
13.	**TEST FOR AMINO ACID** One or two drops of the extract is placed on a filter paper and dried it well. After drying, 1% Ninnydrin is sprayed over the same and dried it well.	Violet colour is formed.	Indicates the presence of **amino acid.**
14.	**TEST FOR ZINC:** The extract is treated with Potassium Ferrocyanide.	No white precipitate is formed.	Absence of Zinc.

INFERENCE: Calcium, tannic acid, unsaturated compound, reducing sugar and amino acid are present in the trial drug Kadukkai Chooranam.

ANNEXURE-III (B)

ANTIMICROBIAL STUDY OF KADUKKAI CHOORANAM

ANTIMICROBIAL ACTIVITY PROCEDURE

Antibacterial Activity Procedure:

Dilution :1mg in 1ml (0.1g in 1ml)

Test Organism:

The test microorganisms used for antimicrobial analysis **Microorganism name** were purchased from Microbial Type Culture Collection and Gene Bank (***MTCC***) Chandigarh. The bacterial strains were maintained on Nutrient Agar (NA) and fungi on Sabouraud Dextrose Agar (SDA).

Nutrient Broth Preparation

Pure culture from the plate were inoculated into Nutrient Agar plate and sub cultured at 37ºC for 24 h. Inoculum was prepared by aseptically adding the fresh culture into 2 ml of sterile 0.145 mol/L saline tube and the cell density was adjusted to 0.5 McFarland turbidity standard to yield a bacterial suspension of 1.5×108 cfu/ml. Standardized inoculum Used for Antimicrobial test.

Antimicrobial Test:

The medium was prepared by dissolving 38 g of Muller Hinton Agar Medium (Hi Media) in 1000 ml of distilled water. The dissolved medium was autoclaved at 15 Lbs pressure at 121^0C for 15 min (pH 7.3). The autoclaved medium was cooled, mixed well and poured petriplates (25 ml/plate)the plates were swabbed with Pathogenic Bacteria culture viz.**Microorganism name** Finally, The Sample or Sample loaded Disc was then placed on the surface of Mullar-Hinton medium and the plates were kept for incubation at 37°C for 24 hours. At the end of incubation, inhibition zones were examined around the disc and measured with transparent ruler in millimeters. The size of the zone of inhibition (including disc) was measured in millimeters. The absence of zone inhibition was interpreted as the absence of activity (Kohner et al., 1994; Mathabe et al., 2006). The activities are expressed as resistant, if the zone of inhibition was less than 7 mm, intermediate (8-10 mm) and sensitive if more than 11 mm (Assam et al., 2010)

ANTI FUNGI ASSAY BY DISC DIFFUSION METHOD (Bauer et al., 1966)

Antibiotic susceptibility tests were determined by agar disc diffusion (Kirby–Bauer) method. Fungi strains (Fungi Name) were swabbed using sterile cotton swabs

in SDA agar plate. Up to 40 µl of each concentration of the extract were respectively introduced in the sterile discs using sterile pipettes. The disc was then placed on the surface of SDA medium and the compound was allowed to diffuse for 5 minutes and the plates were kept for incubation at 22°C for 48 hours. At the end of incubation, inhibition zones were examined around the disc and measured with transparent ruler in milli meters.

Minimum Inhibitory Concentration (MIC) Determination

This assay consists in the determination of chemical agent spectrum of action, according to resistance of studied micro organisms. It was developed the determination of minimum inhibitory concentration (MIC) for every chemical agent, through the classic method of successive dilution. In twelve numbered screw tubes (10 x 100 mm), 1 mL of TSB (trypticase soy broth) medium was distributed for every tube, except for the tube number 1. The tubes were submitted to autoclave under constant pressure and temperature of 121 °C. For the first and the second tubes of the series, 1 mL of tested sanitizing agent was added; tube 2 was stirred and 1 mL was withdrawn and transferred for tube 3. This successive transference was repeated until tube 11. It was added to all flasks, except for flask number

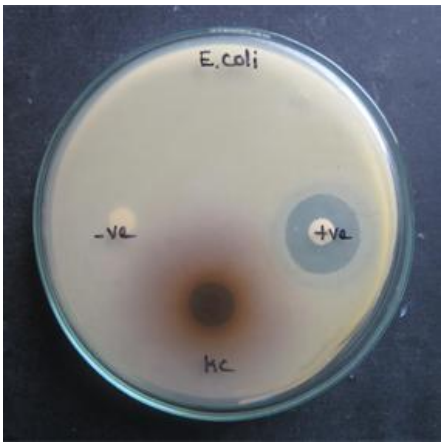
ANTIMICROBIAL RESULTS

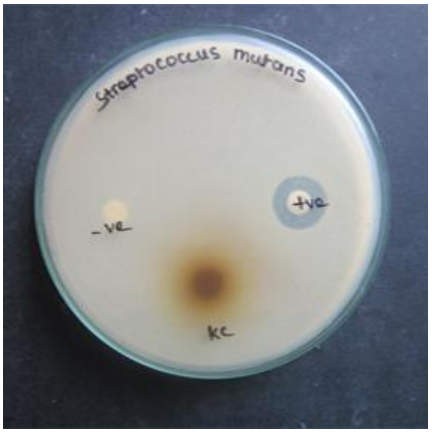
Sample Code	Bacteria Strains Name				
	***Staphylococcus aureus* (G+)**	***Streptococcus mutans* (G+)**	***Bacillus subtilis* (G+)**	***Klebsilla pneumonia* (G-)**	**E – coli (G-)**
KC	**15**	**10**	**13**	**14**	**13**
PC	**28**	**15**	**28**	**28**	**20**
NC	**-**	**-**	**-**	**-**	**-**

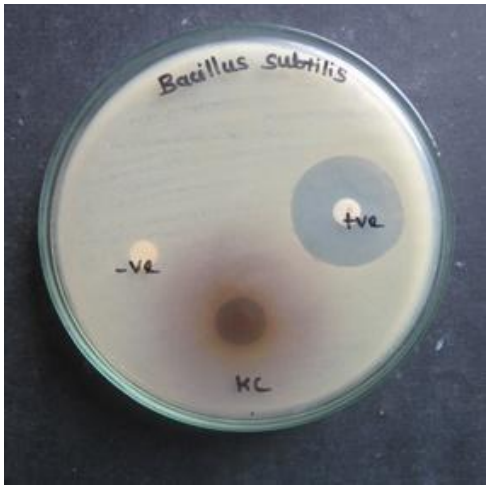
Keys

PC - ***Positive Control (Streptomycin)***
NC - ***Negative Control***
- - ***No Zone***
Mm - ***Millimetre***
G+ - ***Gram Positive Organism***
G- - ***Gram Negative Organism***

Picture: Anti-microbial activity of Kadukkai chooranam







ANNEXURE-IV

BIOSTATISTICAL ANALYSIS OF A PROSPECTIVE OPEN LABELLED NON RANDOMIZED PHASE-II CLINICAL TRIAL OF "KADUKKAI *CHOORANAM*" FOR *AKKINI SELATHUMAM* (DIABETIC NEUROPATHY)"

The statistical analysis for the clinical study is done for 40 cases including both Out- patients and In-patients using SPSS statistical package version 20 was used for data analysis. Comparison between the investigated anthropometric, clinical and laboratory parameters were established using Pearson's correlation coefficient (r). Independent samples t-test (2- tailed) was used to compare means of different parameters. Correlation was significant at the 0.05, 0.01, 0.001 levels (2 tailed).

Descriptive Statistics of FBS (after and before treatment)

	N	Minimum	Maximum	Mean		Std. Deviation
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
FBSB	40	119.00	291.00	189.8250	5.94762	37.61607
FBSA	40	95.00	264.00	153.2750	5.79478	36.64940
Valid N	40					

Descriptive Statistics of PPBS (after and before treatment)

	N	Minimum	Maximum	Mean		Std. Deviation
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
PPBSB	40	191.00	400.00	287.5750	7.54056	47.69072
PPBSA	40	105.00	310.00	213.3500	7.13717	45.13941
Valid N	40					

Descriptive Statistics of PAIN SCORE (after and before treatment)

	N	Minimum	Maximum	Mean		Std. Deviation
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
PainscoreB	40	5.00	9.00	6.8750	.17970	1.13652
PainscoreA	40	1.00	7.00	2.5750	.16751	1.05945
Valid N	40					

Descriptive Statistics of HBA1C(after and before treatment)

	N	Minimum	Maximum	Mean		Std. Deviation
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
HBAICB	40	7.10	10.00	8.7700	.12896	.81562
HBAICA	40	6.80	9.80	8.4575	.13373	.84577

Valid N	40										

Correlations of FBS,PPBS,HBA1C,PAIN SCORE and BMI in study samples (after and before treatment)											
		FBSB	FBSA	PPBSB	PPBSA	HBAIC. B	HBAIC.A	Pain score. B	Pain score. A	BMI.B	BMI.A
FBSB	Pearson Correlation	1	.707**	.642**	.464**	.342*	.402*	.019	.120	.047	.034
	Sig. (2-tailed)		.000	.000	.003	.031	.010	.909	.462	.772	.837
	N	40	40	40	40	40	40	40	40	40	40
FBSA	Pearson Correlation	.707**	1	.520**	.665**	.268	.341*	.043	.243	-.055	-.051
	Sig. (2-tailed)	.000		.001	.000	.094	.031	.794	.130	.734	.754
	N	40	40	40	40	40	40	40	40	40	40
PPBSB	Pearson Correlation	.642**	.520**	1	.692**	.211	.281	.097	.006	-.248	-.245
	Sig. (2-tailed)	.000	.001		.000	.190	.079	.552	.971	.123	.128
	N	40	40	40	40	40	40	40	40	40	40
PPBSA	Pearson Correlation	.464**	.665**	.692**	1	.162	.272	-.125	.051	-.205	-.203
	Sig. (2-tailed)	.003	.000	.000		.318	.089	.444	.755	.205	.209
	N	40	40	40	40	40	40	40	40	40	40
HBAICB	Pearson Correlation	.342*	.268	.211	.162	1	.946**	.101	.166	-.005	-.040
	Sig. (2-tailed)	.031	.094	.190	.318		.000	.535	.306	.974	.805
	N	40	40	40	40	40	40	40	40	40	40
HBAICA	Pearson Correlation	.402*	.341*	.281	.272	.946**	1	.024	.180	.012	-.017
	Sig. (2-tailed)	.010	.031	.079	.089	.000		.885	.267	.940	.918
	N	40	40	40	40	40	40	40	40	40	40
Pain sc ore B	Pearson Correlation	.019	.043	.097	-.125	.101	.024	1	.487**	-.097	-.077
	Sig. (2-tailed)	.909	.794	.552	.444	.535	.885		.001	.550	.635
	N	40	40	40	40	40	40	40	40	40	40
Pain score A	Pearson Correlation	.120	.243	.006	.051	.166	.180	.487**	1	-.034	-.015
	Sig. (2-tailed)	.462	.130	.971	.755	.306	.267	.001		.835	.928
	N	40	40	40	40	40	40	40	40	40	40
BMIB	Pearson Correlation	.047	-.055	-.248	-.205	-.005	.012	-.097	-.034	1	.994**
	Sig. (2-tailed)	.772	.734	.123	.205	.974	.940	.550	.835		.000
	N	40	40	40	40	40	40	40	40	40	40
BMIA	Pearson Correlation	.034	-.051	-.245	-.203	-.040	-.017	-.077	-.015	.994**	1
	Sig. (2-tailed)	.837	.754	.128	.209	.805	.918	.635	.928	.000	
	N	40	40	40	40	40	40	40	40	40	40
**. Correlation is significant at the 0.01 level (2-tailed).											
*. Correlation is significant at the 0.05 level (2-tailed).											

Correlations of LIPID PROFILE in study samples (after and before treatment)											
		TCB	HDLB	LDLB	VLDLB	TGLB	TCA	HDLA	LDLA	VLDLA	TGLA
TCB	Pearson Correlation	1	-.013	.706**	.294	.462**	.292	-.336*	.285	.172	.317*
	Sig. (2-tailed)		.935	.000	.065	.003	.068	.034	.075	.288	.046
	N	40	40	40	40	40	40	40	40	40	40
HDLB	earson Correlation	-.013	1	-.280	-.120	-.240	-.305	.027	-.243	-.156	-.150
	Sig. (2-tailed)	.935		.080	.462	.135	.056	.870	.130	.337	.356
	N	40	40	40	40	40	40	40	40	40	40
LDLB	Pearson Correlation	.706**	-.280	1	-.167	.038	.165	-.112	.002	.064	.236
	Sig. (2-tailed)	.000	.080		.302	.815	.309	.491	.990	.694	.142
	N	40	40	40	40	40	40	40	40	40	40
VLDLB	Pearson Correlation	.294	-.120	-.167	1	.907**	.205	-.265	.302	.129	.313*
	Sig. (2-tailed)	.065	.462	.302		.000	.204	.099	.058	.427	.049
	N	40	40	40	40	40	40	40	40	40	40
TGLB	Pearson Correlation	.462**	-.240	.038	.907**	1	.333*	-.298	.477**	.198	.393*
	Sig. (2-tailed)	.003	.135	.815	.000		.036	.062	.002	.220	.012
	N	40	40	40	40	40	40	40	40	40	40
TCA	Pearson Correlation	.292	-.305	.165	.205	.333*	1	-.007	.585**	.089	.076
	Sig. (2-tailed)	.068	.056	.309	.204	.036		.966	.000	.583	.640
	N	40	40	40	40	40	40	40	40	40	40
HDLA	Pearson Correlation	-.336*	.027	-.112	-.265	-.298	-.007	1	-.073	-.144	-.199
	Sig. (2-tailed)	.034	.870	.491	.099	.062	.966		.655	.377	.218
	N	40	40	40	40	40	40	40	40	40	40
LDLA	Pearson Correlation	.285	-.243	.002	.302	.477**	.585**	-.073	1	.062	.086
	Sig. (2-tailed)	.075	.130	.990	.058	.002	.000	.655		.705	.597
	N	40	40	40	40	40	40	40	40	40	40
VLDLA	Pearson Correlation	.172	-.156	.064	.129	.198	.089	-.144	.062	1	.747**
	Sig. (2-tailed)	.288	.337	.694	.427	.220	.583	.377	.705		.000
	N	40	40	40	40	40	40	40	40	40	40
TGLA	Pearson Correlation	.317*	-.150	.236	.313*	.393*	.076	-.199	.086	.747**	1
	Sig. (2-tailed)	.046	.356	.142	.049	.012	.640	.218	.597	.000	
	N	40	40	40	40	40	40	40	40	40	40

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Changes in Diagnostic findings before and after treatment

Mrs.T.Selvi / 40 Y / Female
IP No 2300

Before Treatment

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MRS.SELVI
Age /Sex : 40 Y / Female
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 16731646
Reg.Date & Time : 10/09/2018 07 : 30
Coll Date & Time : 10/09/2018 08 : 55
Report Date & Time : 10/09/2018 12 : 39

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	174	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**9.23**	%	Non-Diabetic Level:<6.0 Goal :<7.0 UnSatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	218.20	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	206	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	31	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	186	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	100	mgs/dl	Normal :<150 mg/dl Borderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
VLDL CHOLESTEROL Method : Calculation Specimen : SERUM.	12	mgs/dl	10 - 40 mgs/dl
Non-HDL Cholesterol Specimen : SERUM.	160		<160 mg/dl
CHO / HDL RATIO Method : Calculation Specimen : SERUM.	4.3	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM.	2.8	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM.	2.4		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

• TIRUNELVELI : # 177, TVM Road, Vannarpettai , Ph: 0462 - 250 1353, Mobile: 98430 40346.
• PALAYAMKOTTAI : Lakshmi Complex, North High Ground Road, Ph: 0462 - 258 1353 | 258 1363.
• TUTICORIN : # 40, Palai Road, Ph: 0461 - 232 7333 | 232 7353 . Mob: 95401 10515.
• MADURAI : # 4, Dr. Thangaraj Salai, Madurai Ph: 0452 - 252 1353, Mobile: 99402 80527.
• TANJORE : # 22/1, Pudukottai Road, Ph: 04362 - 228 626, Mob: 99420 22448.
• TENKASI : # 242, Samba Street, Ph: 04633 - 223211. Mob: 98421 60517.
• KOVILPATTI : #197, Sandhai Pettai, Ph: 04632 - 228 626, Mob: 99420 22448.
• RAJAPALAYAM : # 64, Kamaraj Nagar, 2nd Street, Ph: 223101. Mob: 99421 10524.

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

After Treatment

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MRS.SELVI
Age /Sex : 40 Y / Female
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 16731544
Reg.Date & Time : 07/12/2018 08 : 30
Coll Date & Time : 07/12/2018 10 : 55
Report Date & Time : 07/12/2018 12 : 45

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	155	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**8.20**	%	Non-Diabetic Level:<6.0 Goal :<7.0 UnSatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	188.65	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	166	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	39	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	111.7	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	110	mgs/dl	Normal :<150 mg/dl Borderline high:150-199 mg/dl High :200-499 mg/dl Very high :>500 mg/dl
VLDL CHOLESTEROL Method : Calculation Specimen : SERUM.	15.3	mgs/dl	10 - 40 mgs/dl
Non-HDL Cholesterol Specimen : SERUM.	127		<160 mg/dl
CHO / HDL RATIO Method : Calculation Specimen : SERUM.	4.3	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM.	2.9	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM.	2.0		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

• TIRUNELVELI : # 177, TVM Road, Vannarpettai , Ph: 0462 - 250 1353, Mobile: 98430 40346.
• PALAYAMKOTTAI : Lakshmi Complex, North High Ground Road, Ph: 0462 - 258 1353 | 258 1363.
• TUTICORIN : # 40, Palai Road, Ph: 0461 - 232 7333 | 232 7353 . Mob: 95401 10515.
• MADURAI : #4, Dr. Thangaraj Salai, Madurai Ph: 0452 - 252 1353, Mobile: 99402 80527.
• TANJORE : # 22/1, Pudukottai Road, Ph: 04362 - 228 626, Mob: 99420 22448.
• TENKASI : # 342, Samba Street, Ph: 04633 - 223211. Mob: 95401 10517.
• KOVILPATTI : #127, Sandhai Pettai, Ph: 04632 - 228 626. Mob: 99420 22448.
• RAJAPALAYAM : # 64, Kamaraj Nagar, 2nd Street, Ph: 223107. Mob: 99401 10524.

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

Nerve Conduction study

Before Treatment

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573759

1175 - MRS.T.SELVI
40 Years / Female / Ht- / Wt-
Patient Summary Ref By: DR.AJANTHAN
Date :09/09/2018 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.50	10.21	7.40mV		
2.Knee	9.79	17.81	9.02 mV	390	52.13

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.54	13.25	3.50 mV		
2.Knee	10.63	18.96	3.30 mV	390	53.67

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.88	11.25	13.37 mV		
2.Popliteal Fossa	10.75	20.63	9.67 mV	360	45.71

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.75	12.25	11.72 mV		
2.Popliteal Fossa	13.38	22.75	7.99 mV	360	41.74

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	2.50	3.30	15.0 µV	140	56.00
2.Mid Calf - Lt	2.55	3.60	18.0 µV	140	54.90

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.50	42.50	45.00	45.00	43.80		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.30	42.90	45.00	44.60	39.60		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance* (mm)	F-Velocity (m/s)
4.00	51.50	55.00	53.50	44.60		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.80	47.90	50.08	49.20	43.60		0.00

RNOTE: The results may be clinically correlated.

After Treatment

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573759

1175 - MRS.T.SELVI
40 Years / Female / Ht- / Wt-
Patient Summary Ref By: DR.AJANTHAN
Date :10/12/2018 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.75	13.75	8.20mV		
2.Knee	9.70	21.35	6.70mV	390	53.03

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.06	13.02	8.02 mV		
2.Knee	9.69	18.33	5.00mV	390	56.84

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.68	11.99	5.56 mV		
2.Popliteal Fossa	11.67	20.10	4.10mV	360	45.51

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.17	12.92	8.60 mV		
2.Popliteal Fossa	11.67	21.56	5.60 mV	360	42.67

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	1.85	2.40	34.20µV	140	75.68
2.Mid Calf - Lt	1.75	2.70	14.40µV	140	80.00

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	49.50	55.50	52.50	47.50		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	45.75	54.75	50.25	43.75		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.25	56.50	63.75	60.13	53.25		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
4.50	51.50	60.25	55.88	47.00		0.00

RNOTE: The results may be clinically correlated.

Changes in Diagnostic findings before and after treatment

Mrs.Chellammal / 54 Y / Female
OP No 44668

Before Treatment

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MRS.CHELLAMMAL
Age /Sex : 54 Y / Female
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 26031556
Reg.Date & Time : 01/11/2018 08:25
Coll Date & Time : 01/11/2018 09:15
Report Date & Time : 01/11/2018 12:55

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	179	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**9.4**	%	Non-Diabetic Level:<6.0 Goal :<7.0 Unsatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	223.08	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	225	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	42.6	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	130.5	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	149.8	mgs/dl	Normal :<150 mg/dl Borderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
VLDL CHOLESTEROL Method : Calculation Specimen : SERUM	30	mgs/dl	10 - 40 mgs/dl
Non-HDL Cholesterol Specimen : SERUM	180		<160 mg/dl
CHO / HDL RATIO Method : Calculation Specimen : SERUM	4.8	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM	3.1	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM	3.5		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

After Treatment

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MRS.CHELLAMMAL
Age /Sex : 54 Y / Female
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 16031560
Reg.Date & Time : 09/02/2019 08:13
Coll Date & Time : 09/02/2019 08:24
Report Date & Time : 09/02/2019 13:02

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	150	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**8.20**	%	Non-Diabetic Level:<6.0 Goal :<7.0 Unsatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	188.64	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	190	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	45.50	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	122.4	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	110.8	mgs/dl	Normal :<150 mg/dl Borderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
VLDL CHOLESTEROL Method : Calculation Specimen : SERUM	22.2	mgs/dl	10 - 40 mgs/dl
Non-HDL Cholesterol Specimen : SERUM	144.5		<160 mg/dl
CHO / HDL RATIO Method : Calculation Specimen : SERUM	4.2	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM	2.7	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM	2.4		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

Nerve Conduction study

Before Treatment

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573739

1175 - MRS.CHELLAMMAL
54 Years / Female / Ht- / Wt-
Patient Summary Ref By: DR.AJANTHAN
Date: 05/11/2018 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.75	12.38	8.66 mV		
2.Knee	11.00	21.50	6.92 mV	380	48.48

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.63	12.25	6.80 mV		
2.Knee	11.88	22.38	5.21 mV	380	43.24

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.63	15.13	11.96 mV		
2.Popliteal Fossa	13.25	27.25	6.93 mV	360	45.71

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.38	13.75	17.52 mV		
2.Popliteal Fossa	13.25	24.38	12.18 mV	360	37.47

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	2.50	3.35	15.0 µV	140	56.00
2.Mid Calf - Lt	2.55	3.60	18.0 µV	140	54.90

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.50	51.75	60.25	56.00	49.25		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.75	45.00	49.75	47.38	42.25		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance + (mm)	F-Velocity (m/s)
3.50	50.25	55.50	52.88	46.75		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.75	53.50	61.00	57.25	49.75		0.00

RNOTE: The results may be clinically correlated.

After Treatment

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573739

1176 - MRS.CHELLAMMAL
54 Years / Female / Ht- / Wt-
Patient Summary Ref By: DR.S.ALAGESAN.ME
Date: 09/02/2019 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.00	11.38	5.67 mV		
2.Knee	9.50	18.88	5.25 mV	390	52.00

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.25	11.75	3.61 mV		
2.Knee	9.50	19.50	2.79 mV	390	53.79

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.88	11.25	13.37 mV		
2.Popliteal Fossa	10.75	20.63	9.67 mV	360	45.71

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.75	13.25	11.72 mV		
2.Popliteal Fossa	11.38	22.75	7.99 mV	360	41.74

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	1.85	2.40	16.2 µV	140	75.68
2.Mid Calf - Lt	1.75	2.20	14.4 µV	140	80.00

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	49.50	55.50	52.50	47.50		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	45.75	54.75	50.25	43.75		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.25	56.50	63.75	60.13	53.25		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
4.50	51.50	60.25	55.88	47.00		0.00

RNOTE: The results may be clinically correlated.

Changes in Diagnostic findings before and after treatment

Mrs.Ramalakshmi / 52 Y / Female
OP No 14898

Before Treatment

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MRS.RAMALAKSHMI
Age /Sex : 52 Y / Female
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 16031560
Reg.Date & Time : 09/02/2019 09 : 09
Coll Date & Time : 09/02/2019 10 : 24
Report Date & Time : 09/02/2019 12 : 20

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	139	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**9.80**	%	Non-Diabetic Level:<6.0 Goal :<7.0 UnSatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	271	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	211	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	44.2	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	138	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl Normal :<150 mg/dl Borderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	144.4	mgs/dl	10 - 40 mgs/dl
VLDL CHOLESTEROL Method : Calculated Specimen : SERUM	28.9	mgs/dl	
Non-HDL Cholesterol Specimen : SERUM.	167		<160 mg/dl
CHO / HDL RATIO Method : Calculated Specimen : SERUM.	4.8	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM.	3.1	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM.	3.3		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

• TIRUNELVELI : # 177, TVM Road, Vannarpettai, Ph: 0462 - 250 1353, Mobile: 98439 43346. • TANJORE : # 22/1, Pudukottai Road, Ph: 04362 - 278544 38804.
• PALAYAMKOTTAI : Lakshmi Complex, North High Ground Road, Ph: 0462 - 258 1353 / 258 1363. • TENKASI : # 242, Samba Street, Ph: 04633 - 223211. Mob: 99401 60517.
• TUTICORIN : # 40, Palai Road, Ph: 0461 - 232 7353 / 232 7363. Mob: 99401 10515. • ERAYILKUTTI : #107, Sandhai Pettai, Ph: 04632 - 228 626. Mob: 99408 23448.
• MADURAI : #4, Dr. Thirugaraj Sale, Madurai Ph: 0452 - 252 1353, Mobile: 99403 80307. • RAJAPALAYAM : # 44, Kamaraj Nagar, 2nd Street, Ph: 223457, Mob: 99401 10504.

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

After Treatment

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MRS.RAMALAKSHMI
Age /Sex : 52 Y / Female
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 17131560
Reg.Date & Time : 11/05/2019 09 : 09
Coll Date & Time : 11/05/2019 10 : 24
Report Date & Time : 11/05/2019 12 : 20

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	129	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**9.23**	%	Non-Diabetic Level:<6.0 Goal :<7.0 UnSatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	218.2	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	166	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	39	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	111.7	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl Normal :<150 mg/dl Borderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	76.3	mgs/dl	10 - 40 mgs/dl
VLDL CHOLESTEROL Method : Calculated Specimen : SERUM	15.3	mgs/dl	
Non-HDL Cholesterol Specimen : SERUM.	127		<160 mg/dl
CHO / HDL RATIO Method : Calculated Specimen : SERUM.	4.3	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM.	2.9	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM.	2		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

• TIRUNELVELI : # 177, TVM Road, Vannarpettai, Ph: 0462 - 250 1353, Mobile: 98439 43346. • TANJORE : # 22/1, Pudukottai Road, Ph: 04362 - 278544 38804.
• PALAYAMKOTTAI : Lakshmi Complex, North High Ground Road, Ph: 0462 - 258 1353 / 258 1363. • TENKASI : # 242, Samba Street, Ph: 04633 - 223211. Mob: 99401 60517.
• TUTICORIN : # 40, Palai Road, Ph: 0461 - 232 7353 / 232 7363. Mob: 99401 10515. • ERAYILKUTTI : #107, Sandhai Pettai, Ph: 04632 - 228 626. Mob: 99408 23448.
• MADURAI : #4, Dr. Thirugaraj Sale, Madurai Ph: 0452 - 252 1353, Mobile: 99403 80307. • RAJAPALAYAM : # 44, Kamaraj Nagar, 2nd Street, Ph: 223457, Mob: 99401 10504.

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

Nerve Conduction study

Before Treatment

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573759

1175 - MRS.RAMALAKSHMI
52 Years / Female / Ht: / Wt:
Patient Summary Ref By: DR.AJANTHAN
Date: 09/02/2019 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.25	15.38	4.23 mV		
2.Knee	12.00	25.38	3.41 mV	390	44.57

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.38	14.63	3.08 mV		
2.Knee	14.75	25.00	2.25 mV	390	37.59

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.75	14.13	12.99 mV		
2.Popliteal Fossa	14.50	24.75	6.85 mV	350	32.49

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.38	14.75	10.06 mV		
2.Popliteal Fossa	12.63	25.13	6.18 mV	350	38.92

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	3.40	4.70	21.0 µV	140	41.18
2.Mid Calf - Lt	3.15	4.05	14.3 µV	140	44.44

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.25	54.25	60.75	57.50	51.00		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.50	57.25	64.00	60.63	53.75		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.00	56.75	65.25	61.00	53.75		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.50	58.25	63.75	61.00	54.75		0.00

RNOTE: The results may be clinically correlated.

After Treatment

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573759

1175 - MRS.RAMALAKSHMI
52 Years / Female / Ht: / Wt:
Patient Summary Ref By: DR.AJANTHAN
Date: 10/05/2019 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.20	11.28	3.67 mV		
2.Knee	9.50	18.18	5.25 mV	400	50.50

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.63	12.23	6.80 mV		
2.Knee	11.88	22.38	5.21 mV	400	43.34

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.63	15.13	11.96 mV		
2.Popliteal Fossa	13.25	27.25	6.93 mV	370	45.71

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.75	12.25	11.72 mV		
2.Popliteal Fossa	13.38	22.75	7.99 mV	370	41.34

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	2.50	3.30	15.0 µV	140	56.00
2.Mid Calf - Lt	2.55	3.60	18.0 µV	140	54.90

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	49.50	55.50	52.50	47.50		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	45.75	54.75	50.25	43.75		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.25	56.50	63.75	60.13	53.25		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
4.80	53.80	61.25	57.25	49.25		0.00

RNOTE: The results may be clinically correlated.

Changes in Diagnostic findings before and after treatment

Mr . Selladurai / 49 Y / Male

IP No 1942

Before

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MR.SELLADURAI
Age /Sex : 49 Y / male
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 16731550
Reg.Date & Time : 30/07/2018 08 : 10
Coll Date & Time : 30/07/2018 09 : 24
Report Date & Time : 30/07/2018 11 : 20

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	179	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**8.0**	%	Non-Diabetic Level:<6.0 Goal :<7.0 Unsatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	207	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	190	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	45.5	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	122.4	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	110.8	mgs/dl	Normal :<150 mg/dl Boderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
VLDL CHOLESTEROL Method : Calculated Specimen : SERUM.	**22.2**	mgs/dl	10 - 40 mgs/dl
Non-HDL Cholesterol Specimen : SERUM.	**144.5**		<160 mg/dl
CHO / HDL RATIO Method : Calculated Specimen : SERUM.	4.2	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM.	2.7	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM.	2.4		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

TIRUNELVELI : # 177, TVM Road, Vannarpettai., Ph: 0462 - 250 1253, Mobile: 99420 40348, 98422 [illegible] 38504.
TENKASI : # 22/1, Pudukottai Road, Ph: 04362 - 222211 Mob: 99401 60517,
TUTICORIN : # 40, Palai Road., Ph: 0461 - 232 7252 | 232 7363 . Mob: 99401 16355,
KOVILPATTI : #197, Sandhai Petral, Ph: 04632 - 228 626, Mob: 99420 22448,
MADURAI : #4, Dr. Thangaraj Salai, Madurai Ph: 0452 - 252 1353. Mobile: 99420 80527,
RAJAPALAYAM : # 64, Kamaraj Nagar, 2nd Street, Ph: 225101 Mob: 99401 18504.

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

The Aarthi Health Care Group : Radiology | Physiotherapy | Endoscopy | Pathology | Cytology | Tissue Culture

After

AARTHI SCANS & LABS
ISO 9001:2015 ORGANISATION

Patient : MR.SELLADURAI
Age /Sex : 49 Y / male
Referrer : GOVT.SIDDHA MEDICAL COLLEGE
Branch : TIRUNELVELI-VPI

SID No. : 18731555
Reg.Date & Time : 10/11/2018 07 : 10
Coll Date & Time : 10/11/2018 08 : 24
Report Date & Time : 10/11/2018 12 : 00

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
GLUCOSE (F) Method : Glucose Oxidase - Peroxidase Specimen : FLUORIDE PLASMA	160	mgs/dl	74 - 106
Glycosylated HbA1c(HPLC)			
HbA1c Method : HPLC	**7.09**	%	Non-Diabetic Level:<6.0 Goal :<7.0 Unsatisfactory control:7.1 - 8.0 Action suggested :8.0
Mean Blood Glucose Level	174	mg/dl	
LIPID PROFILE			
CHOLESTEROL Method : Cholesterol Oxidase,esterase,Peroxidase Specimen : SERUM	155	mgs/dl	<200
HDL CHOLESTEROL Method : Direct Specimen : SERUM	39.2	mgs/dl	35 - 55 mgs/dl
LDL CHOLESTEROL Method : Calculated Specimen : SERUM	111.7	mgs/dl	Desirable level: < 130 mgs/dl Border line : 130 - 159 mgs/dl Elevated : > 160 mgs/dl
TRIGLYCERIDES Method : Lipase/Glycerol Dehydrogenase Specimen : SERUM	76.3	mgs/dl	Normal :<150 mg/dl Boderline high:150-199 mg/dl High :200-499 mg/dl very high :>500 mg/dl
VLDL CHOLESTEROL Method : Calculated Specimen : SERUM.	15.2	mgs/dl	10 - 40 mgs/dl
Non-HDL Cholesterol Specimen : SERUM.	126		<160 mg/dl
CHO / HDL RATIO Method : Calculated Specimen : SERUM.	4.3	Ratio	Optimal<3.5 Goal <5.0
LDL/HDL RATIO Specimen : SERUM.	2.9	Ratio	1.5-3.5
TGL/HDL Ratio Method : Calculated Specimen : SERUM.	2.0		Ideal :<2.0 High risk :>4.0 Very high risk:6.0

TIRUNELVELI : # 177, TVM Road, Vannarpettai., Ph: 0462 - 250 1253, Mobile: 99420 40348, TENKASI : # 22/1, Pudukottai Road, Ph: 04362 - 222211 Mob: 99401 60517,
TUTICORIN : # 40, Palai Road., Ph: 0461 - 232 7252 | 232 7363 . Mob: 99401 16355, KOVILPATTI : #197, Sandhai Petral, Ph: 04632 - 228 626, Mob: 99420 22448,
MADURAI : #4, Dr. Thangaraj Salai, Madurai Ph: 0452 - 252 1353. Mobile: 99420 80527, RAJAPALAYAM : # 64, Kamaraj Nagar, 2nd Street, Ph: 225101 Mob: 99401 18504.

Note : This imaging modality is having its own limitations. Hence this report should be correlated with clinical features and other parameters.

The Aarthi Health Care Group : Radiology | Physiotherapy | Endoscopy | Pathology | Cytology | Tissue Culture

Nerve Conduction study

Before

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573759

1175 - MR.CHELLA DURAI
49 Years / male / Ht: / Wt:
Patient Summary Ref By: DR.AJANTHAN
Date: 15/11/2018 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.00	11.38	5.67 mV		
2.Knee	9.50	10.88	5.25 mV	390	52.00

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.25	11.75	6.31 mV		
2.Knee	9.50	10.50	2.79 mV	390	53.79

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.88	11.25	13.37 mV		
2.Popliteal Fossa	10.75	20.63	9.67 mV	360	45.71

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	4.75	12.25	11.72 mV		
2.Popliteal Fossa	13.38	22.75	7.99 mV	360	41.74

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	1.85	2.40	16.2 µV	140	75.68
2.Mid Calf - Lt	1.75	2.70	14.4 µV	140	80.00

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	49.50	55.50	52.50	47.50		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.00	45.75	54.75	50.25	43.75		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.25	56.50	63.75	60.13	53.25		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
4.50	51.50	60.25	55.88	47.00		0.00

RNOTE: The results may be clinically correlated.

After

Clarity

Dr.ALAGESAN NEURO CENTRE
NO:2,CHATRAM STREET, MURUGANKURUCHI, TIRUNELVELI. PH : 0462 2573759

1175 - MR.CHELLA DURAI
49 Years / male / Ht: / Wt:
Patient Summary Ref By: DR.AJANTHAN
Date: 04/12/2018 Tech. :

MNC Studies

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.75	11.38	8.66 mV		
2.Knee	11.00	21.50	6.92 mV	400	48.48

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	2.63	12.25	6.80 mV		
2.Knee	11.88	22.38	5.21 mV	400	43.24

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.63	15.13	11.96 mV		
2.Popliteal Fossa	13.25	27.25	6.93 mV	370	38.44

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	3.38	13.75	17.52 mV		
2.Popliteal Fossa	13.25	24.38	12.18 mV	370	37.47

SNC Studies

Nerve: Sural R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf - Rt	2.50	3.30	15.0 µV	140	56.00
2.Mid Calf - Lt	2.55	3.60	18.0 µV	140	54.90

F-Wave Studies

Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.50	53.75	60.25	56.00	49.25		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
2.75	45.00	49.75	47.38	42.25		0.00

Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.50	50.25	55.50	52.88	46.75		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
3.75	53.50	61.00	57.25	49.75		0.00

RNOTE: The results may be clinically correlated.

URKUND

Urkund Analysis Result

Analysed Document:	Rajendram Ajanthan.docx (D53947126)
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Registration No: .....

DEPARTMENT OF POTHU MARUTHUVAM

This is to certify that the dissertation topic A Prospective open labeled Non-Randomized phase-II clinical trial on herbal drug **"KADUKKAI CHOORANAM"** for the treatment of **AKKINI SELATHUMAM** (Diabetic Neuropathy) has been approved by the screening committee.

Branch	Department	Name	Signature
I	Pothu Maruthuvam	Prof.Dr.A.Manoharan MD(S)	
II	Gunapadam	Dr.A.Kingsly MD(S) (Associate Professor)	
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26/5/17

Place : Palayamkottai

Date : 26.05.2017

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INSTITUTIONAL ETHICAL COMMITTEE,
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R.No.GSMC/5676/P&D/Res/IEC/2014 **Date: 29.05.2017**

CERTIFICATE OF APPROVAL

Address of Ethical Committee	Government Siddha Medical College, Palayamkottai-627002, Tirunelveli district.
Principal Investigator	**Dr.Rajendram Ajanthan, M.D(s),** First year, Department of PothuMaruthuvam, Reg. No: Not yet registered.
Supervisor & Guide	**Prof.Dr.A.Manoharan, M.D**$_{(s)}$., Head of the Department, Department of PothuMaruthuvam, Government Siddha Medical College and Hospital, Palayamkottai - 627002, Tirunelveli District. drmanoharan25@gmail.com
Dissertation Topic	AProspective open labelled Phase – II Non -Randomized Clinical trial on herbal formulation of ***"Kadukkai Chooranam"*** for the treatment of **AKKINI SELATHUMAM** (Diabetic Neuropathy)
Documents Filed	(1)Protocol (2)Data Collection Forms (3)Patient Information Sheet (4)Consent Form (5)SAE (Pharmacovigilance)
Clinical/Non Clinical Trial Protocol (Others-Specify)	Clinical Trial Protocol-yes
Informed Consent Document	Yes
Any other Document	Case Sheet/Investigation Documents
Date of IEC Approval & its Number	GSMC IV-IEC/2017/Br-I/ 08/29.05.2017

We approve the trial to be conducted in its presented form.

The Institutional Ethical Committee expects to be informed about the process report to be submitted to the IEC at least annually of the study, any SAE occurring in the course of the study, any changes in the protocol and submission of final report.

Chairman

(Prof. Dr. Murugesan M.D(s).,)

Member Secretary

29/5/17

(Prof.Dr.R.Neelavathy MD(s).,Ph.D.,)

K.M. COLLEGE OF PHARMACY – MADURAI

IAEC – CERTIFICATE

This is to certificate that the project title **A PROSPECTIVE OPEN LABELED NON-RANDOMIZED CLINICAL TRIAL OF "KADUKKAI CHOORANAM" FOR AKKINI SELATHUMAM (DIABETIC NEURITIS)** has been approved by the IAEC/RAJENDRAM AJANTHAN/TNMGRMU/MD(S)/321611008/KMCP/19/2018.

Dr. N. CHIDAMBARANATHAN

Name of the Chairman / Member Secretary IAEC:

Dr P. THIRUPATHY KUMARESAN

Name of the CPCSEA Nominee

Signature with Date 11/3/18

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K. M. COLLEGE OF PHARMACY
MADURAI-625 107.

11/3/18

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INSTITUTIONAL ANIMAL ETHICS COMMITTEE
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MADURAI-625 107

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CPCSEA Nominee

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Clinical Trial Details (PDF Generation Date :- Mon, 01 Jul 2019 12:12:03 GMT)

CTRI Number	CTRI/2018/03/012756 [Registered on: 22/03/2018] - **Trial Registered Prospectively**
Last Modified On	21/03/2018
Post Graduate Thesis	Yes
Type of Trial	Interventional
Type of Study	Siddha
Study Design	Non-randomized, Active Controlled Trial
Public Title of Study	A clinical study to study the drug kadukkai chooranam on Neerizhivu
Scientific Title of Study	A prospective open labelled phase II non randomized clinical trial on herbal formulation of "Kadukkai Chooranam" for the treatment of Akkini selathumam (Diabetic Neuropathy)

Secondary IDs if Any

Secondary ID	Identifier
NIL	NIL

Details of Principal Investigator or overall Trial Coordinator (multi-center study)

Details of Principal Investigator	
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Phone	9443886700

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai - 600 032.

This certificate is awarded to Dr/~~Mr/Mrs~~.....RAJENDRAM AJANTHAN.....

for participating as ~~Resource~~ ~~Person~~ / Delegate in the XXIII Workshop on

"RESEARCH METHODOLOGY & BIOSTATISTICS"

Organized by the Department of Siddha,

The Tamil Nadu Dr. M.G.R. Medical University from 6th to 10th March 2017.

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CONTINUING MEDICAL EDUCATION PROGRAMME

Conducted by

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This certificate is awarded to Dr / ~~Mr~~ / ~~Mrs~~ RAJENDRAN...AJANTHAN.....
has participated in the CME Programme held on 05.12.2018 at Conference Hall, Special Therapy Wing, Government Siddha Medical College, Palayamkottai, Tirunelveli. This programme is focused on "HIV / AIDS"

6/12/18

Prof .Dr.A.MANOHARAN , M.D (s) , (Ph.D)
Head , Department of PothuMaruthuvam
Government Siddha Medical College,
Palayamkottai

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Principal
Government Siddha Medical College
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Pre – Siddha Day Seminar on
Scope of Clinical Practice in Siddha System of Medicine

This certificate is proudly presented to Dr/~~Mr/Mrs/Ms~~.....R. AJANTHAN G. TEAM-I 1st PRIZE for Participating / Presenting Poster entitled"..THE COMPARATIVE STUDIES.....HYPERGLYCEMIC ACTIVITY." in the **Pre – Siddha Day Seminar on** "Scope of Clinical Practice in Siddha System of Medicine" organized by Siddha Clinical Research Unit, Palayamkottai, a peripheral unit of Central Council for Research in Siddha(CCRS), Chennai with the support of Ministry of AYUSH held on 19th December 2018 at Govt. Siddha Medical College Auditorium, Palayamkottai.

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Organizing Secretary
Research officer(S) Sci ii i/c
SCRU, Palayamkottai

K. Sivaranjani

Dr K.Sivaranjani
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Siddha Clinical Research Unit
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Original Research Paper

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Ayurveda

ANTI HYPERLIPIDEMIC ACTIVITY OF SIDDHA FORMULATION OF KADUKKAI CHOORANAM IN WISTAR ALBINO RATS

Dr. Ajanthan. R* Department of Pothu Maruthuvam, GSMC, Palayamkottai, Tamil Nadu. *Corresponding Author

Dr. Manoharan. A Professor, Head of the Department, Department of Pothu Maruthuvam, GSMC, Palayamkottai, Tamil Nadu.

ABSTRACT Hyperlipidemia is a metabolic disorder due to various causes, the more prevalence of hyperlipedemia is 80% to 88% (Sarita.M.Kapgate,Abhijit.B.Patil., 2016) with approximately 40% - 48% is more incidence in middle age group. Hyperlipidemia is more in Hepatic disorder and other Endocrine diseases. Objective of the study is to evaluate the therapeutic efficacy and safety profile of Kadukkai Chooranam (KC). The high fat diet induced Wister albino rats , to evaluated by atherogenic index used this study. The results was recorded before and after administration of KC. The results showed KC is a good antihyperlipidemic,it was compared to treated groups and standard groups Is $p < 0.01$.The significant reduction in serum Total cholesterol, triglyceride, LDL, VLDL and moderate increase in HDL level after using Kc. Kadukkai Chooranam(KC) exhibited significant atherogenic index and percentage protection against hyperlipidemic rats. So, Kadukkai Chooranam is affordable cost, highly efficacy and more acceptable, which makes a good choice for lipid control.

KEYWORDS : Kadukkai Chooranam , Hypolipidemic Activity, HFD, Biochemical

INTRODUCTION:

Many people with diabetes have been produced risk factor in atherosclerotic heart disease and diabetic related complications. It may produce high blood pressure, excessive body weight and high blood glucose levels. Dyslipidemia further raises risk of Ischemic heart disease, Peripheral vascular disease and Stroke .In the clinical practice, dyslipidemia patients were associated with diabetes and other non communicable disease ,few patients have suffering in familial hypertriglycedemia. The following drugs available to manage dyslipidemia, statins is a first drug of choice to control hyperlipedemia,folate ,nicotinic acid and bile sequentance are next line of treatment , the above drugs are used long period they have produced Rhabdomylosis , muscular disease and hepatobillary disease .So, newer drug is very essential to treat hyperlipedemia (D.J. Ecobichon,.1997).The following physiological mechanisms have occurred in high cholesterol and diabetic state. The formation and accumulation of advanced glycation products, increased oxidative stress, activation of proteinkinase C pathway, increased activity of hexosamine pathway and vascular inflammation and the impairment of insulin action in the vascular tissue can produced complications.(A.E. Ahire, et al.,2005)

The present study, the effect of siddha formulation of Kadukkai chooranam(KC) is changes in Lipid profiles and blood sugar level.(Gajenda Kumar, et al.,2013)

MATERIALS AND METHODS

The "Kadukkaichooranam" is mentioned in several Siddha literature "Gunapadam Mooligaivaguppu Part-1"is indicated for Mega disorder (Diabetes), Burning sensation of upper and lower limbs(Poly neuropathy), liver diseases and anaemia. In various journal reviwed Terminalia chebula is an Antioxidant (SarmisthaSaha etal,2014), AntiHyperglycemic(NaiamoluKoteswaraRao, 2006)Antimicrobial (Golam MOSTAFA.M et al. 2011) actvities.

The HFD induced Wister albino rats weighted 180±10 animals were used in this experiments. The rats were maintained in accordance with guidelines of the national institute of nutrition, Indian council of medical research, Hyderabad, India and study approved given by Institutional animal ethical committee.

Experimental procedure:

All the animals were weighed and divided into five groups each of six animals,totally 30 rats were used. The details are given below,

Group I : Normal control.

Group II : Cholesterol control. Fed ***cholesterol*** at a dose of400mg/kg body weight for 30 days.

Group III :fed cholesterol as in group II and ***Atorvastatin*** 1mg/kg body weight from days 15 to day 30.

Group IV :fed cholesterol as in group II and siddha formulation Kadukkai chooranam(KC) at a dose of 100mg/kg body weight from days 15 to day 30.

Group V : fed cholesterol as in group II and siddha formulation Kadukkai chooranam(KC) at a dose of 200mg/kg body weight from days 15 to day 30.

At the end of 30 days all the rats were sacrificed, blood was collected and serum was obtained by centrifugation. The serum samples were used for various biochemical studies. The results is correlated by Statistical analysis, ANOVAs and Newmankeuls multiple test. Atherogenic index is followed in study.

Atherogenic index (AI) and LDL-C/HDL-C ratio

- The AI was calculated by the following formula
- AI = (total cholesterol – HDL-C)/HDL-C
- LDL-C/HDL-C ratio was calculated as the ratio of plasma LDL-C to HDL-C Levels

Table 1: Effect of siddha formulation kadukkai chooranam in various Lipid levels Statistical analysis

Table 1: Effect on siddha formulation kadukkai chooranam **in Lipid Profile**

GROUPS	Total cholesterol (Mg/dl)	Triglycerides (mg/dl)	HDL(mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	AI	LDL/HDL
Normal Control	49.70 ± 1.70	57.10± 0.90	26.45 ± 1.18	14.30 ± 0.76	30.86 ± 1.05	0.87 ± 0.50	0.54 ±
Cholesterol Control	115.40 ± 1.56**(a)	161.5 ± 1.68**(a)	11.90 ± 0.65**(a)	30.96 ± 1.32**(a)	13.05 ± 0.70**(a)	8.51 ± 1.33**(a)	2.60**(a)
Standard Control	72.60± 1.42**(b)	82.10 ± 1.80**(b)	21.5 ± 0.40**(b)	20.05 ± 0.76**(b)	24.85 ± 0.76**(b)	2.34 ± 2.33**(b)	0.92**(b)
Treatment control	92.86 ± 1.24**(a)	114.40 ± 1.96**(b)	17.0 ± 0.50**(b)	25.25 ± 0.60**(b)	17.84 ± 0.42**(b)	4.39 ± 1.48**(b)	1.46**(b)
Treatment control	83.85 ± 0.94**(b)	96.5 ± 1.10**(b)	20.30 ± 1.28**(b)	22.12 ± 0.72**(b)	21.25 ± 0.52**(b)	3.13 ± 0.24**(b)	1.08**(b)

After the treatment TC, Triglycerides &LDL also decreased like stranded control group, same time HDL also increased .Values are expressed as Mean ± SEM.Values were find out by using ONE WAY ANOVA followed by Newman Keul's multiple range tests.** (a) values were significantly different from normal control at P< 0.01.** (b) Values were significantly different from hyperlidemic control at P< 0.01. In Table no 1. The end of result found, after the treatment of Kadukkai choornam ,TC, Triglycerides &LDL was decreased in treatment control (Group IV,V),it was compared Group I,II and III standard control .

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Research Article
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ACUTE AND SUB ACUTE TOXICITY STUDY OF SIDDHA DRUG KADUKKAI CHOORANAM (TERMINALIA CHEBULA, RETZIUS)

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ABSTRACT

Background: The Plant is the important source of bioactive compound. The Kadukkai chooranam (*Terminalia chebula* Retzius) has been mentioned in various classical siddha literatures. The text book *Gunapadam Mooligai Vaguppu (Murugesu Mudaliyar C.S, 2013), and Kadukkai, Vallari in thani manbu(Hakeem P.Mohamatu Abthulla Shajabu. 1992)* clearly mentioned in therapeutic effects of Akkini selathumam and it has been similarly correlated symptoms in modern medicine is Diabetic neuropathy. **Objective:** To determined and found the acute and sub-acute toxicity of kadukkai chooranam (KC) on Wister Albino rat. of Kadukkai chooranam (KC) was carried out as per the (OECD)-423 guidelines after getting the confirmation about the animal ethical clearance Committee. In the acute toxicity study were carried out female albino Wister rats, after single oral dose (50 mg/kg) administration of Kadukkai chooranam(Kc), in sub acute toxicity study male and female albino rats were administered daily oral doses (50, 100,200 and 400 mg/kg respectively) of Kadukkai chooranam(KC) for 28 days. At the end of the study, the animals were humanely sacrificed and assessed for the effect of Kadukkai chooranam(KC), assessed by body weight, haematological, biochemical and histopathological investigations. **Results:** In the acute toxicity study no mortality or behavioral changes were observed after using in single dose of Kadukkai chooranam(KC) (2000 mg/kg). indicating that the LD_{50}. **Conclusion**: These results exhibit the absence of acute and sub acute oral toxicity after treatment of Kadukkai chooranam (KC) in rats. However, further clinical studies humans are needed in order to have sufficient safety evidence for its use in humans.

KEYWORDS: Kadukkai chooranam, Siddha Medicine, Toxicity studies, Albino rats.

INTRODUCTION

Siddha medicine is divided into 32 internal and 32 external types of medicine, most of them herbal-mineral combination drugs. The Kadukkaichooranam is an Internal medicine comes under the Chooranam types of Medicines. Kadukkai is a Combretaceae family, which is used in Siddha and Traditional medicine for constipation, chronic diarrhoea, gastric ulcer, gastroenteritis, asthma, cough, dyspnoea, dyspepsia, hemorrhoids, Candidiasis, parasites, malabsorption syndrome, Hepatomegaly, renal calculi, urinary discharge, tumours, skin disease, memory loss, epilepsy, diabetes, cardiovascular disease, anorexia and wounds(Pathartha Guna Chinthamani, 2009) Nadkarni, K.M., 1976).

According to the WHO, the herbal medicines have been defined as those containing plant parts or plant materials in raw state or processed form (Krishnan, K.S., 1998) containing bioactive principles, are to be considered a important form and ensured to follow the Protocol for drug research in traditional system of medicine. The Siddha system of medicine encompasses around 600 medicinal plants is described in siddha materiamedica (Gunapadam Mooligaivaguppu 2016). From the abundant source of herbal preparations in different formulations are practiced in ten decades. So, it must be ensured that the quality of the drugs should not be compromised, the efficacy of the drugs should be maximized, the adverse effects should be minimized when prepared it in a absolute protocol as mentioned by the literatures.

In Tibetan literature was mentioned, the therapeutic uses in part of plant. Root was used for bone disease, Stem for muscular disease, Bark used for skin disease, and Fruits for internal organ related diseases (Pandy GS, Chunekar et al. 1999).

MATERIALS AND METHODS

Collection and Authentication

The matured fruits of *Terminalia chebulla* was collected from native siddha medical shop in Nagerkoil, Tamilnadu. The fruits was identified and authenticated by the Medicinal Botanist and Gunapadam experts at Government Siddha Medical College and Hospital, Palayamkottai – 627002.

International Journal of Reverse Pharmacology and Health Research

Research article

The Spectroscopic Analysis of Siddha Drug Kadukkai Chooranam

Ajanthan. R*[1], Manoharan.A[2]

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[2]Professor, Head of the Department, Department of PothuMaruthuvam, GSMC, Palayamkottai,Tamilnadu, India

Abstract

Background: The Kadukkai is a one of the main ingredient in triphala chooranam ,for the long period kadukkai is used separately and compound preparation was used in siddha system. The various collections of siddha literatures found kaddukkai is a good antioxidant, antimicrobial and Hypoglycemic activities. Kaddukkai choornam (kc) is separately used in siddha system, for treated anemia and diabetic dyslipidemic state. So, standardization of herbal drug kc is very essential to proved the efficacy and avoid toxicity for long term use. In this study attempt is to revalidate the pharmacological process of preparation of kc, which have been discussed about modern spectroscopic characterization and elemental quantification of Kadukkai chooranam(kc).

Objective: To found the morphology and chemical characterization of the herbal plant formulation of Kadukkai Chooranam.

Methods: The KC is determined by qualitative and quantitative modern analytical methods such as phytochemistry, SEM,XDR and FTIR. The analytical study of kc by using SEM, and found the trace elements by applied Energy dispersive X-ray analysis instrumented successfully detect Functional Group of kc through FTIR study .The above study is compared WHO guidance and correlation with the results.

Results: The above results found the minimum and maximum average Particle Sizes between 101 µm to 1115µm in 10 µm view and 107 µm to 1244µm in 100 µm respectively. The further kc is mostly presence of Nitro compounds, alkenes, alcoholic compounds and trace elements like Zinc, Selenium, Calcium, Potassium and Magnesium.

Conclusion: The Kadukkai chooranam is scientifically proved to prolonged usage. All the scientific data showed permissible limitations, which it is correlated by who guidelines. The phytochemical analysis also performed, the results showed kc is contain phenols, flavonoids and tannic acid.s o,kc is safer and for using longer period.

Keywords

Kadukkai, Kadukkaichooranam, Siddha Medicine, Instrument, phytochemistry

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Quick response code

Introduction

Siddha (Gunapadam) Materia medica is classified into three main categories, the preparatory source is received from herbal, mineral and animal origin's. The form of Siddha medicine is divided into 32 internal and32 external types medicine, most of them herbal-mineral combination drugs. The Kadukkaichooranam[1] is an Internal medicine comes under the Chooranam types of Medicines.

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